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**An epidemiological study in General Dental Practice to
better understand the links between periodontal disease
and other common oral conditions**

Dr. Imogen Midwood

A dissertation submitted to the University of Bristol in accordance with the requirements for award of the degree of research Masters in the Faculty of Oral and Dental Sciences.

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Abstract

Objectives: This observational, cross-sectional multicentre study set out to determine the prevalence of periodontal disease, gingival bleeding and gingival recession and their association with tooth wear and dentine hypersensitivity (DH) in National Health Service (NHS) General Dental Practices' (GDPs) across the South West of England. Data was also collected regarding potential underlying risk factors to identify those associated with periodontal disease, gingival bleeding and gingival recession.

Method: Healthy adult volunteers recruited from 28 NHS GDPs in the South West completed a questionnaire and underwent a clinical examination.

Results: Out of the 814 participants recruited to the study, 75.6% exhibited bleeding on probing (BoP) and 28% had evidence of periodontitis (maximum probing pocket depth (PPD) of 4mm or more with bleeding on probing). Recession was observed in 90% of participants, and was more common on buccal surfaces (49.5% of participants) than lingual/palatal surfaces (25.2% of participants). Maximum PPD was strongly significantly correlated with the presence of BoP. For all tooth surfaces, greater probing depths were more common posteriorly in the mouth, scores improving progressively towards the incisors, BoP showed a similar pattern. Gingival recession was also correlated with tooth wear, and occurred most frequently on the buccal aspect of premolar teeth. The majority of participants brushed their teeth at least twice a day (74%) and had attended the dentist or hygienist at least once in the previous 12 months (87%), mainly for routine check-ups and/or a scale and polish (67.2%). In general, the participants self-reported oral health correlated with clinical findings, which showed a self and health aware group of participants. The potentially causal risk factors of periodontal disease were identified as smoking and being male, obesity was also identified but the finding was less significant, interestingly being stressed was inversely related with periodontal disease.

Conclusion: The relatively low incidence of periodontitis, accompanied by good oral hygiene practices, and a strong self-awareness of oral health indicates that this was a well-cared for population. The study supports existing data that smoking, being male and less significantly obesity are risk factors for periodontal disease, yet the presence of stress was inversely related with periodontal disease, all of which require further studies. This finding promotes that good oral hygiene preventative behaviours and regular dental attendance is associated with less periodontal disease and associated comorbidities.

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This dissertation is dedicated to Laura and Al, on my multiple trips to Bristol over the last 2 years you have offered an open door, with a gin and tonic waiting and a bed to sleep in, your friendship and kindness has been immeasurable. I wish you every happiness in your married life and I look forward to making many more memories with you both in time to come!

Author's Declaration

I declare that the work in this dissertation was carried out in accordance with the requirements of the University's Regulations and Code of Practice for Research Degree Programmes and that it has not been submitted for any other academic award. Except where indicated by specific reference in the text, the work is the candidate's own work. Work done in collaboration with, or with the assistance of, others, in indicated as such. Any expressed views in the dissertation are those of the author.

Signed _____

Date _____

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List of abbreviations

ADHS	Adult Dental Health Survey
BEWE	Basic Erosive Wear Examination
BoP	Bleeding on Probing
BPE	Basic Periodontal Examination
CRP	C Reactive Protein
DFTD	Dental Foundation Trainee Dentist
DH	Dentine Hypersensitivity
GBR	Guided Bone Regeneration
GDPs	General Dental Practitioners
ICD	International Classification of Disease
NHANES	National Health and Nutrition Epidemiological Survey
NHS	National Health Service
OHQoL	Oral Health Related Quality of Life
PPD	Probing Pocket Depth
PDF	Portable Document Format
WHO	World Health Organisation

Chapter 1: Introduction

Periodontal diseases are the diseases that negatively affect the supporting tissues of teeth in a person's mouth (Eaton and Ower, 2015). Dental plaque builds up on the tooth surface daily, the process starting immediately after cleaning and if it is not removed frequently and effectively from the tooth surface at the gingival margin, gingivitis occurs, which is inflammation of the gums (Löe *et al.*, 1965). In a disease susceptible host, whose has genetic and epigenetic risk factors, gingivitis can progress to periodontitis. Periodontitis is characterised clinically as periodontal pocket formation and loss of tooth supporting alveolar bone (Eaton and Ower, 2015). If periodontitis is untreated, the degradation of alveolar bone will ultimately lead to tooth loss (Silva *et al.*, 2015) and there can be a negative impact on the quality of life for that individual (Needleman *et al.*, 2004). Periodontal disease is a global burden (Kassebaum *et al.*, 2014), and is the 6th most prevalent disease of mankind, with a severe form affecting 11% of the population. There are a number of known risk factors (Grossi *et al.*, 1995), some of which are modifiable while others are non-modifiable. There are also associations between periodontal disease and systemic disease (Loos, 2016) as well as other oral conditions (West *et al.*, 2013), but more work is needed to confirm the extent of these relationships.

This study seeks to determine the prevalence rates for periodontal disease in people attending routine dental appointments at NHS GDPs in the South West of England, and to examine the relationship of periodontal disease with other oral conditions and potential underlying risk factors. Throughout this project, periodontitis will be referred to as a pocket depth of equal or more than 4mm and severe periodontitis will describe periodontal pocket depths of equal to or more than 6mm (Preshaw, 2015). There are multiple diseases that affect the periodontium (Caton *et al.*, 2018). The main periodontal diseases that I will focus on are dental plaque biofilm induced gingivitis and periodontitis.

1.1 Prevalence of periodontal disease

Periodontal disease includes both the inflammation and destruction of the soft and hard tissues, which support teeth within alveolar bone and if untreated can ultimately result in tooth loss (Chapple *et al.*, 2015). Global figures available from WHO (WHO, 2017) demonstrate that periodontal disease is very common, showing 46% of adults aged 35-44 have at least one tooth with periodontal pocketing of 4mm or more. Furthermore, in 2010, severe periodontal disease was the sixth most prevalent worldwide disease (Kassebaum *et al.*, 2014), this systematic review and meta-regression analysis carried out between 1990 and 2010, showed the global prevalence of severe periodontitis was 11.2%, although figures varied depending on the region with the lowest figure in 2010 recorded for Fiji (3.7%) and the highest for Argentina (21.1%).

1.1.1 Regional variations in prevalence of periodontal disease

A number of studies have confirmed that periodontitis is prevalent in the Western world (Cobb *et al.*, 2009, Kassebaum *et al.*, 2014, WHO, 2017). In prevalence studies periodontitis is often determined by pocket probing depth (PPD) rather than a basic periodontal exam (BPE), with a PPD of 4mm and above most frequently classed as periodontitis (Preshaw, 2015). Surveys to explore the US national prevalence of periodontitis have been taking place regularly since 1960 (Cobb *et al.*, 2009). The results of national surveys carried out between 1960 and 2004 showed an overall reduction in moderate and severe periodontal diseases characterised by PPD's of over 4mm in US adults, but reported prevalence figures fluctuated over this period. When comparing the results of these studies, however, it must be noted that there were some differences in the definitions of clinical periodontitis employed and furthermore, these studies only carried out a partial mouth examination, which may bias results (Cobb *et al.*, 2009). In the most recent National Health and Nutrition Examination Surveys (NHANES) between 2009 and 2012 a full mouth periodontal exam was undertaken, and data was collected from a more representative sample of the population. Using this more accurate survey it was demonstrated that 46% of US civilian adults, aged 30 years or more had periodontitis, with 8.9% having severe periodontitis (Eke *et al.*, 2015). This data is similar to the global averages for the prevalence for periodontitis and severe periodontitis in 35-44 year olds (WHO, 2017).

In Europe, periodontal health has been reported as improving (Kinane *et al.*, 2008), and the number of edentulous adults is declining, a finding that might be explained by the increase in the provision of preventive dentistry (Reich, 2001). However, the prevalence of periodontal disease across Europe is still high, figures available from WHO (WHO, 2017) report that 48% of adults aged 35-44 have at least one tooth with periodontal pocketing of 4mm or more, with figures varying depending on local region. In the UK, the most comprehensive assessment of dental health is the ADHS, which has been undertaken every 10 years since 1968. In the most recent ADHS (2009) it was demonstrated that the number of edentulous adults had declined from 28% in 1978 to 6% in 1990 (Fuller *et al.*, 2011). Similarly, while

some periodontal disease (pocket depth of 4mm or more) was detected in 45% of adults (White *et al.*, 2011), this was decreased from that reported in the 1998 survey (54%), although a probing depth of 3.5mm or more was used in this earlier study which makes the direct comparison of the surveys difficult (Morris *et al.*, 2001). By contrast, the proportion of participants with deeper (6mm+) pocketing increased from 6% in 1998 to 8% in 2009 (White *et al.*, 2011). The increase in the occurrence of severe periodontal disease may reflect an increase in the retention of teeth with periodontal disease, which may have been extracted in previous generations. This is supported by data which suggests, people are living longer and generally healthier disease-free lives (Christensen *et al.*, 2009). Also, a reduction in dental plaque was noted between 1998 and 2009 (White *et al.*, 2012), which again could have aided the retention and prognosis of periodontally involved teeth. The UK prevalence figures of periodontal disease are high and highlight the need to further educate the population to reduce disease prevalence and progression.

There is very little literature available to establish whether periodontal disease is more prevalent in developing countries compared to industrialised countries. WHO data demonstrates a great variance in prevalence figures across all countries, with some European countries reporting figures that are amongst the highest (WHO, 2017). However, it must be noted that the studies on which the WHO data is based on range both in size and age. It is known that developing countries are economically less developed; there is a lower average income and poorer access to medical and dental care especially within rural communities. Where prevalence figures are high in developing countries, it is thought that this is mainly due to the increased plaque levels and presence of calculus, secondary to poor availability of dental care and education (Pilot, 1998). The effect of socio-economic status on prevalence rates for periodontal disease is discussed in more detail below.

1.1.2 The effect of socio economic status on the prevalence of periodontitis

The burden of oral disease is high for the disadvantaged and poor population groups in both developing and developed countries (Petersen *et al.*, 2005), and tooth loss has been shown to be significantly higher in those with lower incomes (Seerig *et al.*, 2015). In the US, the most recent National Health and Nutrition Examination Survey (NHANES) study (Eke *et al.*, 2015) demonstrated that periodontitis was twice as likely to occur in those with the lowest socioeconomic status compared to the highest. It was also shown that prevalence was associated with level of education, adults who had not completed high school having the highest prevalence of periodontal disease. Further, when poverty was assessed, prevalence was highest in the poorest individuals surveyed (Eke *et al.*, 2015).

Similarly, a Brazilian cross sectional study carried out on 1134 12-year old school children demonstrated that there was a high prevalence (96%) of gingival bleeding in children from a lower socioeconomic status whose fathers had a low educational level (Tomazoni *et al.*, 2017). These children were also found to have dental plaque and dental crowding. Another recent study examined 3757

Japanese workers with a healthy periodontium at an initial examination and re-examined them again after 5 years. The results showed that men working in sales or as drivers had an increased incidence of periodontal disease as compared to professionals (Irie *et al.*, 2017). Interestingly, there was no correlation between occupation and the incidence of periodontal disease for female participants. This could be due to that fact that women across the world tend to be more aware and conscious of their general health and they are more likely to seek medical and dental advice compared to men. Alternatively, it could be that the women reviewed in Japan had similar jobs with a smaller variety of professions, so less of a variation was detected.

Overall, these studies show an increased prevalence of periodontal disease in people with lower socioeconomic status

1.1.3 Prevalence of periodontitis with age and an aging population

The periodontal diseases; gingivitis, periodontitis and periodontal disease associated with a systemic disorder, are among the most common diseases affecting children and adolescents (Oh *et al.*, 2002). Gingivitis affects more than 70% of healthy children older than seven years of age (Page & Schroeder 1982). Although gingivitis is common in children, fortunately it is reversible and rarely progresses to periodontitis in childhood. This is supported by a cross sectional study of 1279 participants aged 13 - 65 years in which it was shown that destructive periodontitis was rarely noted under the age of 18 years, but that the incidence increased rapidly between the ages of 19 and 26 years (Cobb *et al.*, 2009). Unfortunately, however, the quality of this data must be questioned, as this study did not establish a case definition for periodontitis or a threshold for the clinical parameters.

In adults, there have been multiple studies which have shown increases in the prevalence of periodontitis with increasing age. Kassebaum *et al.* (2014), detected a steep increase in the prevalence of periodontal disease between the third and fourth decades of life, with the highest incidence occurring at 38 years old. In a study of Turkish adults over 35 it was found that all adults had some form of periodontal attachment loss and that the proportion of those with more than 3 mm clinical attachment loss increased from 43% in 35-44 year olds to 91% in those aged 65+ years (Ilhan *et al.*, 2017). Similarly in the USA has been shown that a high proportion (68%) of over 65 year olds had some periodontitis, and that prevalence continued to increase beyond 50-59 years being significantly higher in adults aged 70-81 years (Eke *et al.*, 2015). In another recent study in Germany, while the prevalence of periodontitis in adults increased significantly with age until age 50-59, thereafter it remained constant at 74% (Holtfreter *et al.*, 2009). Interestingly, in this study while the clinical attachment loss steadily increased with age, probing depth remained constant after the age of 40. This finding may be a result of active periodontal disease causing an increase in the pocket depth and alveolar bone loss, followed by brushing of the unsupported gingivae leading to gum recession and reduced pocket depth but an increase in the attachment loss figure.

This prevalence data supports the findings of others that periodontal disease is a burden on the elderly population (López *et al.*, 2017). It is suggested that the two main dental diseases, periodontal disease and caries should be considered together in an aging mouth, as they can contribute and be causative of each other. The increasing prevalence of periodontal disease with age could be due to the altered neutrophil function and increased production of pro-inflammatory mediators with age which make a host more susceptible to the bacterial challenge in periodontal disease (Preshaw *et al.*, 2004). Additionally, following population improvements in oral health an increase in tooth retention in the aging population has been seen, thus teeth which may have been previously extracted, are now more likely to be retained (Schützhold *et al.*, 2015). This retention of teeth allows the aging population to eat with their own teeth for longer and improves their quality of life, this therefore is beneficial even if the teeth show signs of being periodontally involved. Lastly, periodontal disease has a chronic and cumulative nature, so more periodontally involved teeth are naturally seen in an older population. Even though periodontitis is more prevalent with age, it has been shown that there has been an improvement in general periodontal health of the elderly population over the last few decades (López *et al.*, 2017).

From reviewing the literature available, it can be seen that periodontitis prevalence increases with age, but further research is needed to identify the main reason for this and determine what measures might best help reduce the periodontal burden on the elderly.

1.1.4 Influence of gender on prevalence figures

Several studies suggest that prevalence of periodontal disease may be higher in men than in women. The most recent US NHANES study demonstrated that there was a large difference in the prevalence of periodontal disease between men and women over 30 (Eke *et al.*, 2015), determined using a Centers of Disease Control and Prevention and the American Academy of Periodontology approved definition of periodontitis. The overall periodontitis prevalence figures were reported as 54.9% in men and 37.4% in women, with severe periodontitis figures of 13.3 and 4.7% respectively (Eke *et al.*, 2015). Similarly, in a study of adults aged 30 and over in Brazil it was demonstrated that significantly more men than women had PPD of 5mm or more (Susin *et al.*, 2005). The most recent UK National Dental Health Study (2009), also demonstrated higher prevalence of periodontal disease in men as compared to women, with overall prevalence figures of 47% and 43%, and figures for severe periodontal disease of 10% and 7%, respectively, but these differences were not significant (White *et al.*, 2011). Furthermore, large population studies, which have been controlled for covariates showed a significant difference of more periodontal destruction in males (Shiau and Reynolds, 2010).

Thus, the majority of the data suggests that males are more likely to suffer from periodontal disease than females. There is some evidence from animal studies that suggests that this may be due to an underlying difference in genetic factors (Shiau and Reynolds, 2010). This is further supported by a review which concluded that there is strong evidence that there are gender differences in immune

function, and that the higher levels of inflammatory proteins observed in men might act as a risk factor for periodontal disease (Shiau and Reynolds, 2010). However, it is also known that men are less likely to practice good oral hygiene than women, which may be the reason for the observed gender difference (Furuta *et al.*, 2011). The increased prevalence of destructive periodontal disease that is seen in males, therefore, could be a combined outcome of an increased incidence of poor lifestyle choices as well as genetic predisposition.

1.1.5 Future trends in the prevalence of periodontitis

In 2008 a review was carried out to determine the global trends in the prevalence of periodontitis over the last 30 years (Hugoson and Norderyd, 2008). It was found that there is a very limited number of studies that provide prevalence data over time, although the data indicated a possible trend of a lower prevalence of periodontitis over recent years. Looking forward, a study that considered the views of 113 experts in the field of periodontology, forecast that there will be a stabilisation in the prevalence figures for periodontitis in Europe by 2025 (Madianos *et al.*, 2016).

One of the difficulties in assessing the prevalence of periodontal disease and determining worldwide figures arises from the fact that very few studies provide comprehensive and comparable information on the epidemiology of periodontitis (Papapanou, 1999). Comparing the data from published studies has been difficult as the definition for periodontitis varies greatly between studies and there are methodological and recording disparities with the use of different parameters to assess the severity and extent of periodontal disease (Brown and Löe, 1993). Tonetti, Claffey and European Workshop in Periodontology group C, (2005) recognised the lack of uniformity within the literature with respect to defining a “case of periodontitis”, so they proposed specific case definitions of periodontitis to be used to identify risk factors in periodontitis only. However, the proposal clearly states the criteria were not designed for the assessment of prevalence of periodontitis across populations and/or age groups.

In summary, periodontal disease remains a prevalent disease globally, and in the majority of cases is totally preventable. Periodontal disease is more common with increasing age, and as we are living in an aging population in which tooth retention is improving, it will continue to be a problem. Positively, it has been predicted that the prevalence of periodontal disease will stabilise in Europe over the next 10 years (Madianos *et al.*, 2016), due to improved oral hygiene practices, patient motivation, personal hygiene and knowledge and information.

1.2 Types of periodontal disease

1.2.1 The periodontal classification system and its development in recent years

Classification systems create a framework to study the aetiology and pathogenesis of diseases. In addition, they can aid clinicians in determining the diagnosis, prognosis and best management of conditions that patients present with. An ideal classification system must be simple, easy to understand, easily reproducible and clinically relevant (López and Baelum, 2015).

The International Classification of Disease 10 (ICD-10) is the most recent and commonly used classification of general diseases (WHO, 1990). Within the ICD-10, diseases of the oral cavity are found under diseases of the digestive system; however, this classification system is not commonly used in periodontology because it is not extensive enough to classify diseases of the periodontium. Joseph Fox created the first classification of 'gum disease' in 1806 (Milward and Chapple, 2003). Since then, the classification has evolved. The 1999 Classification of periodontal diseases and conditions was created by experts in the field at the 1999 International Workshop for the Classification of Periodontal Diseases currently remains the most widely used classification system today (Armitage, 1999). However, this version was recently updated by experts at the World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions (Caton *et al.*, 2018), held in Chicago, USA in November 2017.

There are several major changes in the new 2017 classification system of periodontal diseases (Caton *et al.* 2018). Firstly, the previous category of 'Gingival diseases' has been incorporated into a new class of 'periodontal health, gingival diseases/conditions', this class includes the original 2 subclasses for gingival diseases together with a third new sub-class; periodontal health and gingival health. This new subcategory is for those who have been successfully treated for periodontitis, and emphasises that while their condition is stable, a periodontitis patient remains a periodontitis patient for life after therapy. Secondly, the previous categories of 'chronic' and 'aggressive' periodontitis have been combined to reflect the fact that they have one of three pathophysiologies now recognised as periodontitis and will be staged and graded to describe the severity/complexity of disease and the risk of progression. Lastly, a new classification for peri-implant diseases and conditions was developed as this was missing from the previous classification system.

1.2.2 The 2017 Classification of periodontal diseases and conditions

The main classes of periodontal diseases and conditions according to the new 2017 classification system are shown in Table 1.2.

Table 1.2. Classification of periodontal and peri-implant diseases and conditions 2017

Classification	Sub classification
Periodontal diseases and conditions	
Periodontal Health, Gingival Diseases and Conditions	Periodontal health and gingival health
	Gingivitis: Dental biofilm - induced
	Gingival diseases: Non-dental biofilm-induced
Periodontitis	Necrotising periodontal diseases
	Periodontitis
	Periodontitis as a manifestation of systemic diseases
Other Conditions Affecting the Periodontium	Systemic diseases or conditions affecting the periodontal supporting tissues
	Periodontal abscesses and endodontic-periodontal lesions
	Mucogingival deformities and conditions
	Traumatic occlusal forces
	Tooth and prosthesis related factors
Peri-implant diseases and conditions	
	Peri-implant health
	Peri-implant mucositis
	Peri-implantitis
	Peri-implant soft and hard tissue deficiencies

1.2.2.1 Periodontal health, gingival diseases and conditions

The first class of the new classification system 'Periodontal health, gingival diseases and conditions' is subdivided into 3 classes as shown above in Table 1.2.

Dental biofilm induced gingival disease (gingivitis) only occurs in the presence of dental plaque (Löe *et al.*, 1965). It has been shown that the clinical expression of plaque-induced gingivitis can vary (Tatakis and Trombelli, 2004), this is thought to be due to varying physiological and genetic factors between individuals. Plaque induced gingival disease presents clinically as swelling, redness and bleeding of the gingivae (Carvajal *et al.*, 2016). Once diagnosed, the initial management includes oral hygiene instruction, so the patient learns to effectively remove plaque from the tooth surface and may also require liaising with the patients' general medical practitioner to alter any underlying conditions, which could be exacerbating the gingivitis.

Non-dental biofilm induced gingival diseases, such as gingival pigmentation, can occur due to a variety of causes (Caton *et al.*, 2018). The management of these conditions is dependent on the diagnosis and is usually focused on the removal of the causative factor. It is possible for these conditions to present as gingivitis, which is inflammation of the gingivae, secondary to the non-plaque related cause.

Similar to periodontitis, prevalence figures for gingivitis vary depending on what study examiners define as gingivitis. In the UK in the most recent ADHS 54% of participants examined demonstrated gingival bleeding (White *et al.*, 2011). A general trend of an increasing prevalence of gingival inflammation with age has been observed, but again this has been shown to vary dependant on geographical region (Carvajal *et al.*, 2016). For periodontal disease to occur it must have been preceded by gingivitis, however, not all gingivitis progresses to periodontal disease (Löe *et al.*, 1986). The progression of gingivitis to periodontal disease is dependent on the individual genetics, the presence of risk factors and oral bacteria (Tettamanti *et al.*, 2017), as mentioned previously, periodontal disease affects around 50% of the population (Kassebaum, 2014).

The above categories are now joined by another subclass 'periodontal health and gingival health' as part of the larger class that encompasses periodontal health. Periodontal health and gingival health, can be present on either an intact or reduced periodontium, and this subclass allows the inclusion of patients who have a history of periodontitis but who are currently stable (Chapple *et al.*, 2018). It recognises that such patients are in a state of health, but require lifelong supportive periodontal care to prevent disease reoccurrence, and ultimately remain periodontitis patients for life (Caton *et al.*, 2018).

1.2.2.2 Periodontitis

In the new classification, 'Periodontitis' is the second main class and contains 3 subclasses necrotising periodontal diseases, periodontitis and periodontitis as a manifestation of systemic diseases, categorised based on their pathophysiology (Caton *et al.* 2018).

Necrotising periodontal diseases are acute inflammatory periodontal disorders caused by an opportunistic bacterial infection (Horning and Cohen, 1995), and commonly occur in immunosuppressed individuals such as those suffering from HIV (Patton and McKaig, 1998). The class 'necrotising periodontal diseases' includes necrotising gingivitis (NG), necrotising periodontitis (NP) and necrotising stomatitis (NS) (Caton *et al.*, 2018), it has been suggested that these clinical forms are different stages of the same disease process (Herrera *et al.*, 2014).

Periodontitis as a manifestation of systemic diseases describes periodontal disease in which supporting tissues are degraded as a consequence of systemic diseases (Caton *et al.*, 2018). Thus, if an individual has a systemic disease that significantly modifies the initiation and clinical course of periodontal infections, for example Papillon Lefevre Syndrome, which impairs the ability of the host to cope with the bacterial challenge associated with periodontitis, the resulting periodontitis is classified as 'periodontitis as a manifestation of systemic disease' (Albandar *et al.*, 2018). This sub-class does not include Diabetes Mellitus (DM), as DM is a common systemic disease that can have various effects on the course and presentation of periodontitis, but there is no evidence based, clear pathophysiology of how these diseases are linked (Caton *et al.*, 2018).

The third sub-class 'Periodontitis' comprises the 2 classes that were previously termed chronic and aggressive periodontitis and is by far the most common sub-class in this class (Tonetti et al 2018). Periodontitis is a complex bacterial infection that occurs secondary to the formation of a dental biofilm on a tooth surface (Armitage and Cullinan, 2010), the biofilm leads to an inflammatory response due to a dysbiosis of oral pathogens and the emergence of pathogenic strains, combined with a susceptible host there is a loss of periodontal attachment and supporting alveolar bone. Periodontitis presents with a variable microbial presence, bacterial samples taken from patients with periodontitis show a complex microflora supragingivally and subgingival deposits with an increased number of gram negative and flagellated cells (Listgarten, 1976) compared to health. In the 1999 classification aggressive periodontitis was identified as a distinct class of periodontitis as it was perceived that it was necessary to identify the more problematic periodontitis cases that presented early with severe disease and were at a higher rate of progression (Tonetti *et al.*, 2018). However, in practice it was found that the diagnosis of aggressive periodontitis was difficult due to the overlap in criteria between this and chronic periodontitis. Thus, in the new 2017 classification no distinction between aggressive and chronic periodontitis has been made, instead, periodontitis will now be staged and graded. The staging of periodontitis in the 2017 classification is based on the severity of the disease and the complexity of its management, the stages range from I-IV, with a higher number stage representing an increased severity/complexity (Caton *et al.*, 2018). The stage is then described in terms of its extent and distribution, which can be either; 'localised' when it affects <30% of teeth, 'generalised' when it affects >30% of teeth or lastly a 'molar-incisor distribution' (Caton *et al.*, 2018). The grading defines the predicted rate of disease progression and the individuals anticipated response to treatment, this can be modifying risk factors which the patient may have been exposed to and ranges from A (slowest) to C (fastest) (Caton *et al.*, 2018).

There are no prevalence figures available currently, where staging and grading parameters have been used, due to the newness of the 2017 Classification. However, studies that have determined the prevalence of periodontitis generally did not distinguish between chronic and aggressive periodontal disease figures of around 45% being commonly reported (Section 1.1). Due to the newness of the 2017 classification, it is difficult to assess the usage and benefits of the updated classification system, using staging and grading. Utilisation of the new classification system is not without its problems, as it is unlikely that clinical attachments levels will be utilised by general dental practitioners, whom will most likely revert to the use of PPD.

The clinical management of this subclass of periodontitis is described in more detail below.

1.2.2.3 Other conditions affecting the periodontium

As shown in Table 1.2, this category is subdivided into 5 subclasses, systemic diseases or conditions affecting the periodontal supporting tissues, periodontal abscesses and endodontic-periodontal lesions,

mucogingival deformities and conditions, traumatic occlusal forces and tooth and prosthesis related factors.

Dental plaque is generally the initiating factor of gingivitis which can then progress to periodontitis (Lang *et al.*, 2009). However, in the case of some rare diseases, such as neoplasms or tumours, the disease can have a direct negative effect on the periodontal supporting tissues irrespective of the presence of dental plaque; these diseases are classified as 'systemic diseases or conditions affecting the periodontal supporting tissues' (Albandar *et al.*, 2018). In contrast, the sub-class 'Periodontitis as a manifestation of a systemic disease', discussed above exists for systemic diseases which can change the presentation and occurrence of periodontitis, but this occurs secondary to the presence of dental plaque on the tooth surface.

Abscesses of the periodontium can be considered as an extension of other forms of periodontal diseases, but due to challenges in diagnosis and treatment they were allocated their own class (VI) in the 1999 review of classification (Armitage, 1999), and have now been grouped with endodontic lesions (formally class VII) in the 2017 classification (Caton *et al.*, 2018). Periodontal abscesses localised to periodontal tissues are the third most common dental emergency and occur most commonly in patients with untreated periodontal disease and in patients undergoing periodontal maintenance (Herrera *et al.*, 2000). They are clinically characterised by pain, redness and swelling of tissues, occurring typically below the mucogingival line, and if untreated, periodontal abscesses can lead to rapid destruction of tooth supporting structures and cause a patient to become systemically unwell (Herrera *et al.*, 2000).

Mucogingival deformities and conditions around teeth, describes the presence of recession and its associated factors such as gingival biotype, vestibular depth and fraenum effects (Cortellini and Bissada, 2018). Recession is a common condition which is becoming more prevalent as we are living in an aging population, with a recent study in young adults demonstrating that 100% of participants had evidence of recession on at least one tooth (Seong *et al.*, 2018). Risk factors for recession include the presence of gingival inflammation and dental plaque (Mumghamba *et al.*, 2009, Chrysanthakopoulos, 2014). The management depends on the lesion, but ranges from targeted oral hygiene to two staged grafting procedures (Chambrone and Chambrone, 2003).

The sub-class, traumatic occlusal forces, is further sub classified as primary and secondary occlusal trauma and orthodontic forces (Fan and Caton, 2018). Animal studies have shown excessive occlusal forces alone do not cause periodontal loss of attachment, however, when excessive occlusal forces are in the presence of dental plaque a loss of attachment is detected (Erricson and Lindhe, 1984). Hence, treatment should emphasise controlling inflammation which occurs secondary to the presence of bacterial plaque (Polson, 1986).

Lastly, the sub-class 'tooth and prosthesis related factors' includes those tooth and related prosthetic factors that modify or predispose to plaque induced gingival diseases/periodontitis. These are

diagnosed individually and managed accordingly (Ercoli and Caton, 2018), with the main aim of removing any plaque retentive factors which can cause a negative destruction of the periodontal tissues.

1.2.2.4 Peri-implant diseases and conditions

A huge benefit of the 2017 Classification is the inclusion of dental implants, which were previously omitted. Case definitions were developed for the use of clinicians in individual case management as well as for epidemiology population studies (Caton *et al.*, 2018).

Peri-implant health is characterised clinically as the absence of visual inflammation and bleeding on probing (Araujo and Lindhe, 2018), it is possible for health to exist around an implant with either normal or reduced bone support. There is not a defined implant probing depth which suggest peri-implant health is present, due to the variety of implant abutment shapes and sizes and a variation in implant attachment levels.

Peri-implant mucositis is characterised clinically as the presence of visual inflammation and bleeding on probing (Heitz-Mayfield and Salvi, 2018). Evidence has shown that dental plaque is a causative factor of peri implant mucositis, and effective and efficient removal of plaque from the implant surface can reverse this inflammatory process and return the tissues surrounding the implant to health (Jepsen *et al.*, 2015).

Peri-implantitis is a plaque associated pathological disease occurring in the tissues surrounding dental implants, it is characterised by inflammation of the peri implant mucosa and progressive loss of implant supporting bone (Schwarz *et al.*, 2018). Peri-implantitis progresses in a non-linear and accelerating pattern (Derks *et al.*, 2016). Risk indicators for peri implantitis include patients with a history of severe periodontal disease, ineffective plaque removal from the implant surface, smoking, excess cement and the lack of supportive periodontal therapy (Renvert and Quirynen, 2015) as well as full mouth rehabilitations and wear facets on the prosthetic crowns (Dalago *et al.*, 2017). The management of peri implantitis is beyond the scope of this master's project and remains very controversial, however, it includes removing any disease risk factors such as poor oral hygiene, then completing non-surgical, which may or may not be followed by surgical periodontal therapy with the use of regenerative materials (Prathapachandran and Suresh, 2012, Mahato *et al.*, 2016).

Lastly, peri-implant soft and hard tissue deficiencies, identified as the final class of peri implant diseases and conditions can occur when an implant is placed into a boney ridge that is deficient in bone, meaning that the implant is not fully encompassed and integrated in the alveolar bone (Hämmerle and Tarnow, 2018).

1.2.3 Managing periodontitis from a clinical perspective; diagnosis and treatment

When a patient presents with periodontal disease; diagnosing and classifying the periodontal disease can be a subjective task (Deas and Mealey, 2010). Historically and today, the diagnosis and classification of periodontal diseases has been made on their clinical features (Armitage and Cullinan, 2010). To arrive at a diagnosis a clinician needs to consider the age of the patient, the rate of progression of the disease, the pattern of periodontal destruction, the signs of inflammation and finally the levels of plaque and calculus (Armitage, 2010). The basic periodontal examination (BPE) is a tool, which allows clinicians to screen for periodontal disease and guides them regarding patient management. For PPD's obtained using the BPE/WHO probe, scores which fall over 3.5mm and 5.5mm, represented by the black band on the WHO probe, equate to PPD of 4mm and 6mm respectively (Ower, 2016). If the black band ranging from 3.5mm to 5.5mm is encroached on probing but does not disappear, a BPE score of 3 is given, initial therapy is advised to rule out false pocketing prior to diagnosing the disease as periodontitis. It is only when pockets are greater than 5.5mm (BPE score 4) or when a BPE Score 3 is present after resolution of inflammation, that a full PPD chart is required, periodontitis is diagnosed and root surface debridement is indicated.

Periodontal therapy consists of improving patients' oral hygiene, creating an oral cavity which is cleansable by the patient, non-surgical and surgical therapy to control the bacterial infection, which may or may not include the use of antibiotics and following this, supportive periodontal therapy and maintenance (Pastagia *et al.*, 2006). The maintenance of good periodontal outcomes after an adequate therapy is delivered depends on the quality of the supragingival plaque control (Angst *et al.*, 2017). The dual approach of regular, self-directed, effective oral hygiene alongside professional removal of sub gingival plaque and deposits is usually very effective at controlling the inflammatory process of periodontal disease (Drisko, 2001). However, inter-individual variation in response to periodontal therapy can be varied, and the reasons for this variation in response are not yet clearly understood (Deas and Mealey, 2010).

In summary, a classification system aids clinicians at an individual and population level to study the aetiology, pathogenesis and treatment of periodontal diseases to optimise patient care.

1.3 Pathogenesis

Pathogenesis is a sequence of events leading to the occurrence of a disease (Heasman, 2009). The occurrence of periodontitis is preceded by gingivitis, which is preceded by the formation of a dental plaque biofilm on the tooth surface (Preshaw *et al.*, 2004). However, it should be remembered that as described above, gingivitis may be caused by other factors, and not all lesions of gingivitis progress to periodontitis (Löe *et al.*, 1986).

The pathogenesis, histology, clinical presentation and relationship of the biofilm, gingivitis and periodontitis are described below.

1.3.1 Gingivae in health

Dental plaque is a microbial community that develops on tooth and mucosal surfaces in health (Marsh and Devine, 2011), with the bacterial composition of the biofilm dictated by the oral surface on which it forms (Aas *et al.*, 2005).

The formation of dental plaque on tooth surfaces follows an orderly sequence of events (Marsh and Devine, 2011). The first event is the formation of the acquired pellicle, a conditioning film in which proteins derived from saliva or crevicular fluid are laid down on a newly cleaned or newly exposed tooth surface (Hojo *et al.*, 2009). A subset of planktonic bacteria present in saliva or crevicular fluid interact with binding proteins in the pellicle by weak and reversible electrostatic or physical interactions (Kolenbrander *et al.*, 2002, Huang *et al.*, 2011). Following initial attachment, stronger irreversible interactions form between the adhesins on the microbial cell surface and receptors present in the acquired pellicle (Kolenbrander *et al.*, 2002). Only a small number of species of oral bacteria have properties that enable them to associate with the conditioning film in the first instance. Streptococci spp make up 60-90 % of the early colonisers (Nyvad and Kilian, 1987, Hojo *et al.*, 2009). Other early colonisers include oral species of the genera *Veillonella*, *Prevotella neisseria*, *Gemella* and *Actinomyces* (Dentino *et al.*, 2013).

In the next stage of biofilm formation (co-aggregation), bacterial species that cannot directly colonise the tooth surface, such as *Fusobacterium nucleatum*, bind to receptors on the surface of the early colonisers already attached to the acquired pellicle (Hojo *et al.*, 2009) increasing the microbial diversity within the developing biofilm. The majority of bacterial strains can only co-aggregate with a limited number of partners, and *F. nucleatum* is an important bacteria species as it can interact with a large number of other strains frequently acting as the bridge between early and late colonisers (Kolenbrander *et al.*, 2002). Once attached directly or indirectly to the tooth surface microbes produce polymers such as glucans, which form a plaque biofilm matrix (Marsh, 2010) this matrix acts as a scaffold which hosts molecules and microbes within the dental plaque.

In health the resident oral microbes have a symbiotic relationship with the host (Marsh and Devine,

2011). The biofilm matrix creates a stable microbial community, which protects itself from the harsh oral environment and host response, and protects the host from other pathogenic species (Dentino *et al.*, 2013, Marsh and Devine, 2011). In healthy gingivae at a cell level, the surfaces of the junctional epithelium, sulcus epithelium and oral epithelium are continuous (Preshaw, 2015), creating a selective and protective barrier against bacteria. However, the majority of biopsies of clinically healthy gingivae show low levels of inflammatory cells, present to protect the host against the daily build-up of bacterial plaque and other substances on the tooth surface (Page, 1986). This low level of inflammation may be a direct beneficial result of the oral biofilm in health, as in the gut it has been shown that the biofilm in homeostasis triggers a low level inflammatory response which contributes to the health of the gut (Neish, 2009). Clinically, gingival tissues in health are pink in colour and the free gingival margin has a well-defined knife-edge appearance, lying closely adjacent to the enamel surface (Preshaw *et al.*, 2004) and the levels of gingival crevicular fluids are low (Marsh and Devine, 2011).

1.3.2 Early disease; gingivitis

The ability of plaque to cause gingivitis was first confirmed in a Danish study in 1965 (Löe *et al.*, 1965). Recognised stages of gingivitis have been now identified which describe the progression of the gingivae from health through initial, early and established gingival lesions (Page, 1986). As the quantity and diversity of microbial plaque accumulates, initial gingivitis develops in 0-4 days (Heasman, 2009). Microbial substances and by products, for example, lipopolysaccharides are released and cross the junctional epithelium to enter the gingival tissues and stimulate the cells in the gingival tissues to release inflammatory mediators that cause an inflammatory response (Preshaw *et al.*, 2004). Histologically, an increase in vascular dilation and permeability, an increased flow of gingival crevicular fluid and an increased number of neutrophils passing via the junctional epithelium to the gingival sulcus is seen (Page, 1986), as well as an initial breakdown of collagen fibres around blood vessels.

After 4-7 days of dental plaque presence, an 'early' gingival lesion is formed as a result of the host response to the presence of the biofilm (Payne *et al.*, 1975). The marginal gingival connective tissue becomes infiltrated by inflammatory cells, which are mainly macrophages and T lymphocytes (Page, 1986). As inflammation persists, neutrophils are evident and there is an associated loss of collagen. Concurrently within the gingival connective tissue, the increased presence of host defence cells leads to pathogenic alteration of fibroblasts (Page, 1986), which in turn reduces the fibroblasts' capacity of collagen formation (Heasman, 2009). There is vasodilation and increased permeability of blood vessels, which allows the movement of fluid and defence cells within the gingival tissues (Preshaw *et al.*, 2004). Defence cells can pass towards the gingival sulcus via a gradient to fight the bacterial plaque and its products. If plaque is undisturbed, an early inflammatory lesion lasts up to 21 days, described clinically as chronic gingivitis (Heasman, 2009). The gingival tissues appear red and inflamed, with a swollen and rolled rounded appearance of the gingival margin. Plasma cells predominate, a large number of

neutrophils remain in or near the junctional epithelium (Page, 1986), and there is continued collagen depletion and degradation of the junctional epithelium (Heasman, 2009).

As disease progresses from health to gingivitis there is a change in the host-microbe relationship from a symbiotic one where the microbes live as commensal organisms to a pathogenic host-microbe one (Berezow and Darveau, 2011). This change in host-biofilm relationship is accompanied by a change in the bacterial composition of the biofilm in which the proportion of gram positive bacteria falls and that of gram negative bacteria rises. Analysis of a study by Moore and Moore (1994) by Kolenbrander et al (2006) demonstrated that while 9 out of the top 10 most common bacteria are gram positive in health, this falls to 5 out of the top 10 in gingivitis.

1.3.3 Later disease; periodontitis

The lesions and stages of gingivitis are confined to the gingival tissues. As indicated previously gingivitis does not always progress to periodontitis, it is possible for a state of chronic gingivitis to remain indefinitely (Page, 1986). Thus, the progression of periodontitis not only requires the presence of pathogenic microbes, it must also occur in a susceptible host, and is influenced by factors such as variations in inflammatory response, diabetes and genetics (Offenbacher, 1996). Periodontitis is diagnosed when the chronic inflammatory response to plaque progresses beyond the gingivae, resulting lesions exhibiting an apical migration of the junctional epithelium and alveolar bone destruction.

In susceptible individuals' progression to periodontitis occurs when the host inflammatory response to plaque creates an environmental change within the plaque biofilm which alters its composition to favour periodontal pathogens. For example, a temperature rise of 2°C caused by gingival inflammation (Fedi and Killoy, 1992), favours the pathogens *P. intermedia*, *A. actinomycetemcomitans* and *P. Gingivalis* (Haffajee *et al.*, 1992). This and other changes such as an alteration in pH and available nutrients are thought to result in a shift in the microbial profile of the biofilm towards the anaerobic and proteolytic microbes of periodontal disease, which are able to tolerate the new environment (Marsh and Devine, 2011). Physical contact between microbes allows the binding of more pathogenic late colonisers such as *Tannerella Forsythia*, to biofilms containing *F. nucleatum* (Kuramitsu *et al.*, 2007), and the release of small signalling molecules help pathogenic plaque bacteria regulate their behavior in response to the changing environment (Mahajan *et al.*, 2013). Additionally, as the biofilm matures, microbes communicate through the exchange of genetic information (Dentino *et al.*, 2013), and horizontal gene transfer between bacteria in the biofilm can convert avirulent pathogens to virulent pathogens (Mahajan *et al.*, 2013).

Sigmund Socransky, developed the idea of bacterial 'complexes' which are made up of a collection of microbes, which are commonly found clustered together in varying states of health and disease (Socransky *et al.*, 1998). The complexes are colour coded, periodontal disease is associated with microbes in the orange and red complexes. The orange complex includes; *A. actinomycetemcomitans*

Prevotella intermedia and *F. nucleatum* (Dentino *et al.*, 2013). The gram negative, anaerobic periodontal pathogens, associated with periodontal pockets and bleeding on probing, *P. gingivalis*, *Tannerella forsythia* and *Treponema denticola* form the 'red complex'. Within the red complex, *P. gingivalis* is a key periodontal pathogen (Hernández *et al.*, 2011); its growth can be promoted in the presence of *F. Nucleatum* and *T. Forsythia*, from growth factors that they secrete. It is also involved in cross feeding, *P. Gingivalis* metabolises products released by *T. Denticola*, and in turn releases isobutyric acid which promotes the growth of *T. Denticola*, making it a very virulent and persistent pathogen.

In a healthy mouth in the presence of plaque, the innate and adaptive immune systems come into play, generating an infiltration of inflammatory cells into the periodontal connective tissues to protect the periodontium which is followed by a period of host tissue repair (Cekici *et al.*, 2014). However, when plaque persists and there is a failure to return to homeostasis, the inflammatory and immune response takes over and the subsequent chronic inflammation leads to tissue destruction and disease (Cekici *et al.*, 2014). Plaque deposits are more likely to survive and remain undisturbed by external physical factors in areas of the oral cavity that are crowded (Chung *et al.*, 2000) or sheltered such as the gingival crevice and periodontal pocket (Gomes-Filho *et al.*, 2011, Silva *et al.*, 2015). Areas of dental crowding have shown an increased number of periodontal pathogens in the subgingival microflora (Chung *et al.*, 2000), hence, a patient is more likely to have a periodontal pocket in an area that is more difficult to clean. The tissue destruction seen in periodontal disease is due to a combination of factors; plaque bacteria release substances which cause direct injury to the periodontal tissues and the host immune response to the prolonged presence of pathogenic bacteria releases inflammatory mediators that also cause destruction of the host tissues (Heasman, 2009). Historically, in periodontitis, it was thought that the periodontal destruction was a continuous process (Socransky *et al.*, 1984). However, the most accepted model of bone destruction accepted today is the 'burst theory', where there are times of activity and regression at different oral sites in an individual (Hernández *et al.*, 2011).

The relationship between the microbes, host response and modifying factors is complex, the extent and severity of periodontal disease being determined by the extent of the host response to the bacterial challenge (Hajishengallis and Korostoff, 2017). It can be seen that the pathogenesis of periodontal disease is an extremely complex sequence of events, which includes the formation and dysbiosis of a plaque biofilm on a tooth surface, which can initiate a self-destructive inflammatory response in the periodontal tissues of a susceptible host.

1.4 Problems associated with periodontal disease

Periodontal disease affects individuals over a range of ages all over the globe (Kassebaum *et al.*, 2014). There are many aspects of periodontal disease, which can cause a variety of problems both at an individual and population level, and impact on quality of life (Needleman *et al.*, 2004).

1.4.1 Periodontal disease and its impact on an individual

1.4.1.1 Day to day effect of living with periodontal disease

Periodontal disease should not be regarded as a silent disease; research has found an association between clinically diagnosed periodontal diseases and a reduced oral health related quality of life (OHQoL), OHQoL decreasing with increasing disease severity/extent (Buset *et al.*, 2016).

Similar results have been found in other studies, in a cross sectional study the OHQoL of 205 patients' who were attending a private periodontal clinic was examined (Needleman *et al.*, 2004). Patients reported that their periodontal disease negatively affected their comfort (19%), resulted in halitosis (18%), detracted from their appearance (18%) and worried them (15%) or affected their mood, happiness or comfort (12%) (Needleman *et al.*, 2004). The study also showed that low OHQoL scores were associated with higher numbers of teeth with periodontal pocket depths of 5mm or more and new patients or patients undergoing treatment, as compared to patients in the maintenance phase. This study was limited as there was no control group and only patients receiving private periodontal care were incorporated. In a similar yet larger study carried out in Hong Kong, 767 participants with no/low periodontal attachment loss or high/severe attachment loss completed the Chinese version of Oral Health Impact Profile (OHIP-14S) questionnaire and their scores were compared. It was demonstrated that OHQoL was poorer in the higher clinical attachment group across all measures which included functional limitation, physical pain and disability, psychological discomfort and disability, social disability and handicap, differences that were statistically significant for many of the measures (Ng and Leung, 2006). These studies strongly support the negative impact of periodontal disease on quality of life.

A specific condition associated with periodontal disease, secondary to clinical attachment loss and the loss of supporting alveolar bone around teeth is recession and tooth root exposure, which can cause DH (West *et al.*, 2013). The impact of root sensitivity on OHQoL in periodontal patients was determined in a study carried out in Hong Kong that recruited patients aged 18-35 undergoing supportive periodontal treatment with periodontally stable dentitions, that had undergone treatment at least 6 months prior to the study. Sensitivity was assessed by patient self-report and measured clinically using air blast and tactile stimuli, patients also completed the Chinese Oral Impact on Daily Performances (OIDP) questionnaire. The general trend of the data showed a larger (worse) OHQoL score correlated with a

positive clinical examination score of root sensitivity, but the relationship was weak. Interestingly, the data showed that the impact of sensitivity on OHQoL increased with age (Goh, Corbet and Leung, 2016).

Periodontal disease is also associated with halitosis (Heasman, 2009), an oral malodour, that affects 30-50% of the population. In a recent study, participants with halitosis showed a statistically significant increased number of sites with BOP and PPD's compared to the control group with no halitosis (Lu *et al.*, 2017). This study further confirmed the relationship between halitosis and periodontal disease, however, halitosis could also be due to other oral factors such as dental caries, tongue coating or gut bacteria (Lu *et al.*, 2017). It has been shown that halitosis can result in personal discomfort and emotional stress. When participants in a study in Tanzania were asked about their self-reported halitosis and their OHQoL, a relationship between halitosis and OHQoL was seen (Kayombo and Mumghamba, 2017), confirming the negative impact of the occurrence of halitosis on the functional and psychosocial aspects of quality of life.

As clinicians, it is easy to review clinical parameters to assess disease, without remembering the effects of the disease from the patients' perspective. The assessment of OHQoL is a means by which we can measure and appreciate the effects of periodontal disease on our patients, from their perspective (Sischo and Broder, 2011). Interestingly, however, a study carried out in Canada reviewing the OHQoL in seniors and pre-seniors, showed a higher score for pre-seniors compared to seniors even though the oral health from a clinical examination perspective was better for the pre-senior cohort (Kotzer *et al.*, 2012), a finding that demonstrates how subjective self-perceived oral health as a result of each individuals' expectations and experiences.

1.4.1.2 Systemic effect of periodontal disease at an individual level

The research into the associations between periodontal disease and systemic disease is described as 'periodontal medicine' (Stamm, 1998). Periodontal disease has been associated with fifty-seven systemic conditions (Loos, 2016), some of which include, cardiovascular disease, diabetes, rheumatoid arthritis, cancer, respiratory diseases and adverse pregnancy outcomes. Some systemic conditions, such as diabetes have shown a bidirectional relationship with periodontal disease such that a patient with periodontal disease has an increased risk of diabetes and a patient with diabetes has increased risk of periodontal disease (Casanova *et al.*, 2014), and treating one condition may influence the outcome of the other condition.

Even though many disease associations have been long established, in order for the causal relationship between the diseases to be confirmed, more studies are needed using strict criteria alongside a universal definition of periodontal disease (Linden and Herzberg, 2013). Three potential mechanisms by which periodontal disease could result in systemic diseases have been identified (Van Dyke and van Winkelhoff, 2013). The first mechanism hypothesises that bacteria from the oral cavity invade the periodontal tissues through large ulcerated subgingival periodontal surfaces, then enter blood vessels

eliciting an infection at a distant site, secondary to the bacteraemia. In the second potential mechanism, the inflammatory response to the bacterial accumulation on the tooth surface and in the gingival tissues causes the release of inflammatory mediators which then have an impact on systemic inflammation. Alongside this, the release of harmful by products by invading bacteria may also induce vascular pathology in the circulatory system. The third mechanism postulates that as the adaptive immune response is stimulated in response to the persistent inflammation in the periodontal tissues, T cells release cytokines which contribute to periodontal tissue destruction as well as inflammatory conditions systemically such as diabetes and cardiovascular disease through immunological injury.

The strongest links between periodontal disease and systemic disease have been found for cardiovascular disease and diabetes, these will be described briefly below. Cardiovascular diseases occur secondary to atherosclerosis of blood vessels, and encompass a wide range of conditions from hypertension to myocardial infarction (Eaton and Ower, 2015). Periodontal disease has been found to be associated with carotid atherosclerosis (Zeng *et al.*, 2016), and severe periodontal disease has also been associated with increased aortic arch atheroma thickness and calcification (Sen *et al.*, 2017), which can lead to recurrent vascular events such as stroke and transient ischaemic attacks. Patients with chronic periodontitis show an increased serum level of systemic inflammatory markers such as C reactive protein (Gomes-Filho *et al.*, 2011) and inflammatory cytokines such as IL-1 and IL-6 (D'Aiuto *et al.*, 2004), even after controlling confounding factors. Inflammatory markers such as CRP and IL-6 have also been found to be predictors of peripheral atherosclerosis and its progression (Tzoulaki *et al.*, 2005). Following non-surgical periodontal therapy in patients with severe generalised periodontitis, the levels of systemic inflammatory markers IL6 and CRP were significantly reduced (D'Aiuto *et al.*, 2004), thus treating periodontal disease could play a role in decreasing heart disease initiation and progression. However, there is not enough evidence available to indicate whether periodontal treatment can prevent or treat atherosclerosis at this point in time (D'Aiuto *et al.*, 2013).

Diabetes is a metabolic disease brought about by impaired insulin secretion or insulin action, which presents as hyperglycaemia (Blair, 2016), and is divided into Type 1 and Type 2. The incidence and severity of chronic periodontitis is more common in diabetic patients (Löe, 1993), especially in the presence of hyperglycaemia (Botero *et al.*, 2012). Oral examinations of newly diagnosed diabetics or pre-diabetics showed that both conditions are associated with periodontal pathology, pathology being worse in those already diagnosed, suggesting that in the presence of dysglycaemia all teeth in the mouth are at risk of periodontal disease (Lamster *et al.*, 2014). The mechanism by which periodontal disease may promote diabetes, similarly to cardiovascular disease, is thought to be a result of inflammatory molecules released in response to periodontal disease diffusing into the systemic circulation (Colombo *et al.*, 2012). The chronic inflammation of periodontal disease causes the release of excess reactive oxygen species which could activate pro-inflammatory pathways that promote insulin resistance, but there is still little evidence on the direct causation of diabetes (Taylor *et al.*, 2013). There has been

speculation and a small number of studies carried out over the last 20-30 years to investigate whether treating periodontal disease in diabetic patients can have a positive impact on glycaemic control. To help determine the true outcome a large multi centred randomised control trial was carried out in the USA which concluded that non-surgical periodontal therapy did not improve glycaemic control in patients with diabetes mellitus presenting with moderate to advanced chronic periodontitis (Engelbreton *et al.*, 2013).

In conclusion, the evidence above supports that the presence of chronic inflammation in periodontal disease, is associated with a number of debilitating systematic diseases. A complexity of studying periodontal medicine for researchers, is the potential impact of confounding factors such as smoking which may underlie the disease associations (D'Aiuto *et al.*, 2004), and makes it hard to establish causality. It would seem prudent for clinicians to treat patients' holistically, not only considering their periodontal status, but also medical factors which could influence or be influenced by their periodontal condition. Through informing patients' and making them aware of these associations, patients may be more motivated to take responsibility and work towards improving their oral health.

1.4.1.3 Effect of treatment of periodontal disease on an individual

Initial non-surgical periodontal therapy has been shown to significantly improve oral health related quality of life and well-being (Saito *et al.*, 2010), particularly through reducing pain, difficulties encountered when eating, and gingival bleeding of sufferers of periodontal disease (Vatne *et al.*, 2015). Following initial therapy tooth longevity has been shown to be increased, but a key factor to the long term success of the treatment is continued supportive periodontal therapy and associated patient compliance (Checchi *et al.*, 2002, Lee *et al.*, 2015). Successful treatment of periodontal disease, very importantly relies on the lifelong commitment to changes in lifestyle choices, such giving up smoking (Alexandridi *et al.*, 2017) and maintaining excellent oral hygiene (Meyle and Chapple, 2015) which may be challenging for a patient to commit to. Furthermore, not all risk factors for periodontal disease are modifiable, and patients with non-modifiable risk factors have a lifelong susceptibility to periodontal disease resulting in the need for structured and regular (2-3 monthly) supportive periodontal therapy which could be costly to the individual in both time and money.

Unfortunately, a frequent side effect of periodontal treatment is post-operative sensitivity (Lin and Gillam, 2012). Post-operative sensitivity depends on the type of treatment received, and studies are difficult to compare as different methods of investigating sensitivity are utilised (Lin and Gillam, 2012). While periodontal therapy is beneficial overall to health, patients may experience negative side effects of periodontal therapy, although a systematic review reports a decreased root sensitivity around two weeks after periodontal therapy (Von Troil *et al.*, 2002).

In conclusion, the overarching aim of periodontal therapy is to maintain the health of the periodontium and extend tooth longevity. However, there are some negative effects of undergoing periodontal therapy, many of which are subjective to an individual.

1.4.1.4 Periodontal disease and its impact on a population

Periodontal disease is a prevalent global disease (Kassebaum *et al.*, 2014), which effects many individuals in our population. In 2010, the global economic burden of oral disease was \$442 billion, which was comprised of direct treatment costs as well as indirect costs such as productivity loss (Listl *et al.*, 2015). The global cost of productivity loss due to severe periodontal disease is thought to be \$54 billion/year (Listl *et al.*, 2015). This could have a negative impact on the workforce in terms of the man power and moral.

In addition to costs associated with loss of productivity and treatment, the association of periodontal disease with many other systemic diseases (Loos, 2016), a link that is suggested to be causal in some cases at least, points to a greater impact on the population in terms of overall health. Whether the prevention and treatment of periodontal disease can positively influence any of these systemic diseases is not yet confirmed, but it is likely that improving periodontal health could lead to a healthier population. Applying a health message and a common risk factor approach to a population, such as healthy gums do not bleed, promoting non-smoking and maintaining a healthy diet, will not only positively influence oral health but also many other chronic conditions which share the same risk factor (Sheiham and Watt, 2000).

With time and research more information is becoming available regarding which oral health interventions are most cost effective at a population level (Hettiarachchi *et al.*, 2017), once determined, these will be used to improve oral health of the population.

1.5 Risk factors associated with periodontal disease

Risk factors are characteristics or exposures to an individual that do not cause a disease, but change the likelihood of a disease occurring (Eaton and Ower, 2015). Thus, the identification of risk factors is important as where it is possible to modify them it is possible to reduce the likelihood of disease. There are multiple risk factors for periodontal disease, some of which are more established than others, these fall into 2 categories, modifiable and non-modifiable.

1.5.1 Modifiable risk factors

Modifiable risk factors can be influenced by either the patient and/or the clinician, the main modifiable risk factors for periodontal disease being poor oral hygiene. The association of poor oral hygiene and the presence of periodontal disease has been long established (Greene, 1963) and in a classic study it was showed that the cessation of simple oral hygiene measures in patients with clinically healthy gingivae resulted in gingivitis (Löe *et al.*, 1965), and vice versa, that when oral hygiene is re-established. The link between oral hygiene, gingivitis and the subsequent development of periodontitis was confirmed in a longitudinal study which examined individuals in Sri Lanka and Norway, aged between 14 and 31 years at the commencement of the study. The early data collected showed that Sri Lankan tea labourers who had received no education on the prevention or treatment of dental diseases all scored positively for plaque and gingival bleeding. By contrast the Norway cohort who had a large exposure to good dental care had lower plaque and bleeding scores (Löe *et al.*, 1978). Between 1969-1975 a six-fold increase in tooth loss in the Sri Lankan tea labourers compared to the Norway population was observed which was due to periodontal disease as carious lesions were largely absent. In a follow-up assessment approximately 16 years later, a small number of individuals from the same cohort were examined again. While the majority had progressed to periodontal disease and consequent tooth loss, interestingly, 11% of the Sri Lankan tea labourers demonstrated resistance to periodontal disease exhibiting almost no bone loss, despite gross plaque and calculus deposits on their teeth (Löe *et al.*, 1986). This shows that poor oral hygiene and supragingival plaque removal is a risk factor for periodontal disease but not directly causal in all cases, some patients regardless of their plaque levels remain resistant to periodontal disease.

There are multiple local risk factors in a dentition which have the ability to initiate and propagate periodontal disease through their plaque retentive nature (Eaton and Ower, 2015). Examples of localised plaque retentive factors include; iatrogenic factors such as removal partial dentures or ill-fitting margins of dental crown and bridge work, or anatomical abnormalities of the tooth structure or dental calculus. Through retention of a dental biofilm on these plaque retentive factors, a localised inflammatory response is initiated.

Smoking has also been shown to be a risk factor for periodontal disease, with a 2.7 fold increase of moderate to advanced periodontal disease being demonstrated in smokers as compared to non-

smokers in a case controlled study of 405 patients (Haber and Kent, 1992). Furthermore, data has demonstrated that there is a dose dependent relationship between the number of cigarettes smoked per day and the likelihood of suffering from periodontitis (Heitz-Mayfield, 2005). A study carried out in New York showed that there was a higher risk of severe bone loss in smokers compared to non-smokers, and that this risk increased with the amount smoked, light smokers having a 3.25-fold risk of severe bone loss yet heavy smokers having a 7.28 fold risk (Grossi *et al.*, 1995). Furthermore, a systematic review concluded that there is evidence to show that smokers have a poorer response to periodontal treatment, with smaller pocket depth reduction observed as compared to that seen in non-smokers, although there was some heterogeneity in the results of the studies included (Labriola *et al.*, 2005). Taken together, this data suggests that smoking is most certainly a risk factor of periodontal disease, but to date the mechanism is not fully known. It is thought that smoking has a negative impact on the hosts inflammatory and immune responses to bacterial plaque, and that smoking induced vasoconstriction of the periodontal vasculature may inhibit healing (Palmer *et al.*, 2005).

Stress is described as inadequate response to constant adverse stimuli (Goyal *et al.*, 2013), psychosocial stressors can either be major stressful life events or minor daily stressors or hassles. Stress, and more importantly an individual's ability to cope with an adverse life event can influence the presentation of periodontal disease (Genco *et al.*, 1999). In a large study of 1426 participants; after necessary adjustments of the data, financial strain was indicated as a factor that was associated with increased attachment and alveolar bone loss, especially if the participant had inadequate coping mechanisms (Genco *et al.*, 1999). In a small pilot study, where emotional intelligence, a measure of persons ability to cope, was assessed when periodontal therapy was commenced it was demonstrated that a reduction in plaque scores and bleeding scores was associated with more resilient emotional intelligence (Gamboa, *et al.*, 2005). This study also indicated that there is a relationship between stress and periodontal disease but the nature of the relationship and whether lower emotional intelligence leads to periodontal disease or vice versa is not clear. Thus, this data suggests an association between stress and periodontal disease, however, a direct causal relationship has not yet been demonstrated so the true mechanisms of this association remain unknown (Goyal *et al.*, 2013).

Research into the link between diet and periodontal disease is relatively new, but the majority of cross sectional studies to date have failed to find significant associations between specific nutrients and periodontitis (Dang *et al.*, 2014). However, a 14 year observational study has demonstrated that men with high wholegrain consumption were 23% less likely to develop periodontitis than those consuming minimal levels of wholegrain (Genco *et al.*, 2005). Furthermore, it has recently been found that a deficient intake of micronutrients; such as Vitamin A, B1, C and E, iron, folate and phosphorus are associated with an increased severity of periodontal disease (Luo *et al.*, 2018). There is also much interest in the nature of the link between low Vitamin D levels and the presence of periodontal disease, however the current data available is inconclusive. A number of cross-sectional studies have provided

evidence suggesting an association between low levels of vitamin D and poor periodontal health, but to date there is no evidence that reduced vitamin D levels are causal for periodontitis (Pinto *et al.*, 2018). Interestingly, many European adults have been found to be Vitamin D deficient in the winter months (Cashman and Kiely, 2014), this requires further investigation.

Obesity, which is characterised by a body mass index ratio of 30 or above occurs secondary to the abnormal or excessive deposition of fat in the adipose tissue (Jagannathachary and Kamaraj, 2010), and is a syndromic risk factor for many diseases such as hypertension and type 2 diabetes. It is thought that adipose derived cytokines are released which have the ability to secrete other pro-inflammatory cytokines and molecules which drive the inflammatory process of periodontal disease, increasing levels of periodontal tissue destruction (Genco *et al.*, 2005). Interestingly, the link between obesity and periodontal disease, may be modulated by the level of insulin resistance (Genco *et al.*, 2005), with the presence of periodontal disease being more common in an obese population with a higher insulin resistance. A systematic review of 57 studies concluded that type 2 diabetes could be considered a risk factor for periodontitis but was unable to draw the same conclusion for (non-modifiable) type 1 diabetes (Chávarry *et al.*, 2009). Furthermore, it has been demonstrated that the poorer the glycaemic control is in a diabetic person, the more severe and extensive the presentation of periodontal disease is (Lim *et al.*, 2007). This data confirms that type 2 diabetes at least, is a risk factor for periodontitis and that the severity of disease is likely linked to the degree of glycaemic control.

The data presented here demonstrates that a wide range of modifiable risk factors have been associated with and may influence the presentation of periodontal disease. However, for many, more studies are required to confirm their casual nature for periodontal disease and the mechanisms by which they act. These factors are modifiable by choices of either the patient or the clinician.

1.5.2 Non-modifiable risk factors

As described previously (Section 1.1), the prevalence of periodontitis is more common with increasing age and in males. The rate and effect of aging is specific to an individual but may be influenced by modifiable factors such as lifestyle and environment as well as genetics (Lamster *et al.*, 2000). Similarly, the increased prevalence of periodontal disease in males is not likely to be limited to genetic factors, but may also be due to modifiable factors such as a tendency for poorer oral hygiene, a lower reluctance to seek medical help (Furuta *et al.*, 2011).

Certain individuals have a genetic trait which increases their susceptibility to periodontal disease (Eaton and Ower, 2015). In a classic twins' study which collected data from monozygous and dizygous twins it was demonstrated that there was a significant genetic component associated with the presence of gingivitis, increased probing depth and increased attachment loss (Michalowicz *et al.*, 1991). The data showed that between 38% to 82% of the population variance for the periodontal measures could be attributed to genetic factors. In addition, there was no difference in periodontal disease occurrence in

twins reared both together or apart, suggesting that genetics played a larger role than environmental factors for the presence of periodontal disease. A genetic influence on the presentation of diseases of the periodontium is particularly seen in periodontal disease that progresses rapidly (Vieira and Albandar, 2014).

The identification of non-modifiable risk factors for periodontitis underline the importance for all clinicians treating patients with periodontal disease to take a thorough history as part of the examination process, as there are many known factors which are associated with and thought to be causal for periodontitis. However, it is likely that there are other risk factors that are currently unknown, and further work to identify these is important for the prevention of periodontitis.

1.6 Relationship of periodontal diseases with other oral conditions

Periodontitis is strongly associated with poor oral health (Löe *et al.*, 1978). Thus, it would be expected that periodontal disease would be inversely related to oral conditions such as tooth wear and DH which are generally associated with good oral hygiene. However, gingival recession is a characteristic of periodontitis has been shown to occur in individuals with both good and poor levels of oral hygiene (Cortellini and Bissada, 2018), suggesting the relationship may be more complex.

Tooth wear is a prevalent condition, 76% of individuals displayed some form of tooth wear in the last ADHS in 2009 (White *et al.*, 2011) and in the UK prevalence seems to be increasing; between the ADHS in 1998 and the ADHS 2009 moderate tooth wear increased from 11 to 15% (White *et al.*, 2011). Tooth wear is a multifactorial condition (Lopez-Frias *et al.*, 2012) which can be caused by erosion, abrasion, attrition or a combination of these (Mair, 1992). Erosion is tooth surface wear which occurs secondary to an acidic challenge (Lussi and Carvalho, 2014). The erosive challenge can be intrinsic where the origin of the acidic challenge is from within in the body, generally stomach acid, which reaches the oral cavity due to acid reflux or vomiting resulting in loss of enamel on the palatal surfaces of maxillary teeth (Bartlett *et al.*, 1996). The erosive challenge can also be extrinsic (Manaf, 2012), where the acid originates from external dietary sources, in this instance tooth wear is most commonly observed on the buccal aspects of teeth. In a relatively recent meta-analysis the consumption of soft drinks including fruit juices was shown to be associated with increased tooth wear in the young (Salas *et al.*, 2015).

Abrasion and attrition are associated with physical tooth wear (Barbour and Rees, 2006). Abrasion is tooth wear from foreign object such as a toothbrush, however normal twice daily toothbrushing alone is not sufficient to cause tooth wear (Addy and Hunter, 2003). Attrition occurs from tooth to tooth contact, most commonly being seen on occlusal surfaces in those with bruxism (Rees and Somi, 2018). Erosive challenges play an important role in exacerbating physical tooth wear (Bishop 1997, Lussi and Carvalho 2006), as erosion not only causes tooth surface loss on its own, but also softens enamel

leaving it vulnerable to physical wear (Shellis and Addy, 2014). A number of indices have been developed over the years to assess tooth wear, but many were complex and studies using different indices were difficult to compare as what was scored varied, this led to the development of the Basic Erosive Wear Examination (BEWE) (Bartlett *et al.*, 2008). The BEWE measure is a simple tool used to assesses the severity and localisation of the tooth wear on the buccal, occlusal and lingual tooth surfaces. The BEWE score can either be used as a screening tool and the highest figure from each sextant is recorded or individual to each tooth.

Tooth wear is important to diagnose early, as it is a cumulative and irreversible condition which may lead to a negative aesthetic, and a need for complex restorative work (Peutzfeldt *et al.*, 2014). Tooth wear can also lead to DH. DH is the short sharp pain which occurs secondary to a stimulus such as cold, that cannot be attributed to by any other form of pathology (West *et al.*, 2014). DH occurs when dentine becomes exposed and dentine tubules are opened from the oral cavity to the pulp; fluid within the dentine tubules moves in response to the stimulus eliciting the pain response (West *et al.*, 2014). Tooth wear and gingival recession are the primary causes of DH in the absence of pathology (West *et al.*, 2014). However, dentine is also exposed in periodontal disease, and periodontal treatment can also give rise to sensitivity in root dentine, although this sensitivity is not classed as DH due the presence of pathology (West *et al.*, 2013). DH is a prevalent condition a large epidemiology study in Europe discovered 41.9% of participants reported suffering from the condition (West *et al.*, 2013), and has been shown to adversely affect oral health-related quality of life (Bekes *et al.*, 2008).

While many studies have examined the prevalence of periodontal disease, tooth wear and DH, few have sought to confirm their relationship. However, a recent study focussing on gingival recession in 349 young adults in the UK demonstrated that maximum recession scores correlated positively with maximum DH scores, and clinically relevant tooth wear, as well as pocket probing depths and bleeding on probing (Seong *et al.*, 2018), suggesting a link between gingival recession and all other oral conditions. In another study, looking at a female cohort in Tanzania (Mumghamba *et al.*, 2009), the main factors positively associated with gingival recession were the presence of bleeding on probing and dental calculus.

Current data suggests that recession, tooth wear and dentine DH are linked, however, more work to determine the nature of any relationship between these clinical conditions and periodontitis is needed, as well as further studies to identify potential risk factors and confirm if they are causal.

1.7 Aim of the study

As described earlier in this introduction, periodontal disease is an extremely prevalent condition affecting people throughout the UK and the world (Kassebaum, 2014), which negatively affects quality of life, so there is a global need and relevance to further knowledge in this field. As previously indicated, data regarding the prevalence of periodontal disease varies between studies, making the analysis of general trends of prevalence overall, by gender, by socioeconomic status and by location inconclusive and difficult to assess. The last national UK adult health survey, focussed on periodontal diseases, caries and tooth wear was carried almost 10 years ago. No attempt to determine associations between the clinical conditions was made, and limited data on risk factors for dental diseases was collected, questions in the survey focussing on oral hygiene, smoking and sugar consumption. The present study aimed to provide up to date information on the prevalence of periodontal disease in a similar number of individuals to the ADHS in the South West of the UK. In addition, information about the prevalence of other oral conditions including recession, tooth wear and DH was obtained and associations with periodontal disease determined. This is important to investigate, as there is limited research available about the relationship of periodontal disease with other oral conditions. More comprehensive data about lifestyle habits that may be risk factors for periodontal disease was collected. For the purpose of this study only relationships between potential risk factors and periodontal conditions were examined.

Principal aim

To determine the prevalence of periodontal disease, and identify associated risk factors in adults aged 18 and over, attending NHS GPs in the South West of England.

Secondary aims

In this population:

- To determine the prevalence of recession, tooth wear and DH.
- To investigate the relationship between periodontal disease, recession, tooth wear and DH.
- To investigate the association between patient reported and clinically determined oral conditions.

Specific objectives

- To determine the prevalence of periodontal disease, gingival recession, tooth wear and DH in adult NHS patients by clinical examination and recording of probing depths, the presence or absence of bleeding on probing, recession depths, basic erosive tooth wear examination score and DH as measured by Schiff score.
- To assess the relationship between periodontal disease, gingival recession, tooth wear and DH using appropriate statistical test of association.
- To identify risk factors that are potentially causal for periodontal disease by means of a patient reported questionnaire.

- To assess the relationship between patient reported and clinically determined oral conditions using appropriate statistical test of association.

Hypotheses

- The prevalence of clinical conditions in this population will be similar to that observed in the most recent ADHS (2009).
- Significant positive associations will be seen between periodontal conditions and between tooth wear and DH, but tooth wear and DH will be negatively correlated with periodontal conditions.
- Periodontal disease detected clinically will be significantly positively associated with patient reported poor oral hygiene practices, smoking, obesity and stress.
- Patient reported measures of oral health will be significantly associated with all clinically reported conditions.

Chapter 2: Methods

2.1 Study design

The study was a cross-sectional, observational, epidemiological, multi-centre study which included healthy adult volunteers from the South West of England in 28 different general dental practices which provided NHS dentistry, from Gloucester to Truro. All the dental practices were practices taking part in the Dental Foundation Training scheme, and the data was collected by Dental Foundation Trainee dentists (DFTDs) who had been trained by this investigator and research site staff. Healthy adult volunteers who were attending routine dental examinations at their regular dental practice were approached to take part in the study and the eligibility criteria for the volunteers were minimal enabling the study to be as inclusive as possible. However, there were some exclusions, predominantly of factors which could have adversely affected the data recorded, such as volunteers who required antibiotic cover. The study was approved by the HRA and the North West-Preston NHS Research Ethics Committee (REC; reference 16/NW/0850), sponsored by the University of Bristol and carried out according to GCP (Good Clinical Practice) standards.

2.2 Recruitment of dental foundation dentists, training and calibration

DFTDs in the South West of England now undertake research or audit as part of their training. The present study was offered as one of 3 options, and 28 trainees chose to participate in and collect data for this study. Capacity and capability were confirmed from each of the NHS practices prior to the start of the study in line with HRA requirements.

The DFTDs attended a study training day at the University of Bristol Dental Hospital (BDH) on Thursday 2nd February 2017. On arrival at BDH, the DFTD's were given an individual study information folder specific for their practice which included copies of all the study documents and a copy of the training day presentations. The morning consisted of lecture-based teaching which included an Introduction to Dental Research and Good Clinical Practice Training and a presentation about the aims of the study with advice about how to go about initiating and orchestrating this study in their individual dental practices. The dental foundation trainees were introduced to all the study paperwork of which they had been provided in hard copy (in their study folders) and electronically. The importance of each document was explained, and the order in which they were to be presented and completed by the DFTD for each participant was described. The final presentation reviewed the clinical form and how to score the oral conditions of interest. This lecture consisted of multiple clinical pictures and tooth wear models. Some dental school are not taught the Basic Erosive Wear Index (BEWE) tooth wear index in the undergraduate curriculum, so it was necessary to train all individuals for completeness, to ensure all the dentists had an equal and full knowledge of how to use this BEWE index. At the end of the morning, any questions the foundation dentists had were answered. All the dental trainees had successfully completed a dental degree and had a basic knowledge of the information which was to be gathered so

the information given was mainly provided as a refresher and to make sure all trainees had the same information at the start of the study.

In the afternoon, the DFTDs had a clinical session on the Adult Dental Health Clinic at BDH in order to be calibrated. Sixteen adult volunteers of all ages, who had varying degrees of periodontal status and tooth wear but otherwise healthy, were examined. Volunteers for the training day were provided with a volunteer information sheet ahead of the study day and gave written informed consent prior to the clinical examination. Initially each volunteer was assessed clinically by two dentally qualified staff members (the author of this thesis and examiner experienced in these indices), scores were agreed and the clinical data about two teeth were recorded and kept hidden from the trainees. The DFTDs were then invited into the clinic in pairs, one acted as the clinician and the other as a nurse to record the clinical exam scores on the two selected teeth for each of the 16 volunteers. After each assessment, the clinical scores recorded by the trainees were marked and compared to the scores recorded by the trainer, and discrepancies in results were discussed. During this calibration exercise if a trainee score was different to the agreed examiner score following discussion with the examiners and further training the trainee was asked to score the same condition in another volunteer. This was repeated if necessary until their score(s) matched those given by the examiner. By the end of the clinical session, all the DFTDs has been calibrated and were collecting data to the same standard. At the end of the day, there was a concluding meeting and any questions the DFTDs were answered. The data obtained from the volunteers at BDH was for training purposes only and was not used in this study.

The examiners were informed on the training day, of the equipment needed to carry out the participant examination, which included a dental chair with a 3-in-1 spray, basic periodontal examination (BPE) / World Health Organisation (WHO) probe and Williams periodontal probe. Also, the examiners were advised the patient must not be anaesthetised from a dental injection at the time of the examination.

2.3 Participant recruitment

Due to the variation in practice management and general running of individual dental practices, during the training day held at Bristol Dental Hospital the DFTDs were advised to discuss how to best facilitate participant recruitment at their practice with their educational supervisor. The study aimed to recruit volunteers who were attending for routine dental examinations, potential participants were identified by an appropriate dental practice member who routinely has access to their dental records for the purpose of their dental care. Across the majority of dental practices, patients' due a routine dental examination were asked by the dental practice receptionist or dental nurse whether they were interested in participating in the study and were provided with the patient information sheet. Participants were given ample time to fully read the participant information sheet and ask any questions they had before informed written consent was taken by the DFTD, from the participants who were happy to take part. Participants who consented to take part in the study were then assessed for eligibility by the DFTD, according to the inclusion and exclusion criteria (Table 2.1).

Table 2.1 Study inclusion and exclusion criteria

Inclusion Criteria	Exclusion Criteria
a. Healthy volunteers of either gender who are attending a general dental practice for a check-up or review appointment with a dental professional.	a. Patients attending a general dental practice for a check-up or review appointment who had unstable systemic disease, such as uncontrolled diabetes or cardiovascular disease.
b. Aged 18 or over.	b. Aged under 18.
c. Understand and are willing, able and likely to comply with all study procedures and restrictions.	c. Do not understand study procedures and restrictions and are unlikely to comply with them.
d. Accept the form of the study and sign a declaration of informed consent.	d. Do not give informed consent to participate in the study.
e. Have a minimum of 10 teeth not including implants or crowns or bridges	e. Have less than 10 teeth.
f. Able to respond to the questionnaire questions.	f. Persons incapable of responding to the questions.
g. Not employees of the general dental practice or family/relative of the employer.	g. An employee of the general dental practice, and/or a family relative of the employee.
h. Have not used analgesic medication/drugs or received/applied topical anaesthetic in the preceding 4 hours.	h. Persons who have used analgesic drugs or had used a topical analgesic in the preceding 4 hours
i. Do not require antibiotic cover (following infectious endocarditis, using prosthetic cardiac valves).	i. Persons who require antibiotic cover (following infectious endocarditis, using prosthetic cardiac valves).
j. Suitable to take part in the study.	j. Anyone who in the investigator's opinion is not suitable to take part in the study.

2.4 Data collection

After enrolment into the study participants were asked to complete the study questionnaire (Appendix 1). This questionnaire was a modified version of that used in the European study by West *et al.* (2013). Questionnaires were completed in the dental surgery waiting rooms, during completion of the questionnaire, the DFTD was available to answer any questions and assist with the questionnaire as necessary. The questionnaire took around 5-10 minutes to complete and asked for demographic data, such as age, gender, socioeconomic status and level of study achieved. It also collected data on the participant's oral hygiene practices and data about the participant's oral health quality of life experience. Lastly, it asked for information on the evaluation of risk factors associated with periodontal disease, tooth wear and DH. These included tobacco usage, medication, diet, the participant's history of seeking aid from the dental care system, health associated preventive behaviours as well as their weight and height.

Following completion of the questionnaire, the DFTD completed a clinical examination of the entire dental arch excluding the wisdom teeth, tooth recordings were taken buccally and lingually/palatally

and recorded on a data collection sheet (Appendix 2). Periodontal conditions included the participant's periodontal pocket probing depths (mm), recession (mm) and the presence and absence of gingival bleeding were recorded. Erosive tooth wear was recorded using the BEWE (0=no erosive wear to score 3=wear with tissue loss on more than 50% of the surface, Appendix 2), with the localisation of the lesion (coronal, root surface, crown-root junction) also recorded. The presence of exposed dentine was recorded as either visible dentine or no visible dentine, while DH was determined following an air blast using the examiner scored Schiff index (0=no sensitivity to 3=continuous painful sensitivity on stimulus) (Appendix 2) and patient reported response (0=no DH, 1=yes DH). Dental implants and teeth with crowns and bridges were excluded from the analysis.

The BPE was recorded with a standard BPE/WHO probe. During data collection, the periodontal pocket probing depths, recession and bleeding presence were examined at 6 points on every tooth using a Williams probe, and the highest number of either the three buccal or three lingual/palatal surfaces were recorded. The periodontal pocket probing depth was measured from the base of the periodontal pocket to the gingival margin and recession was recorded from the cemento-enamel junction to the gingival margin, both of which were measured with a Williams probe. For the tooth wear scores and dentine exposure the buccal or lingual/palatal individual tooth surfaces were looked at as a whole and recorded for data analysis.

To make data collection as simple and complete as possible for each DFTD, a plastic wallet containing all the necessary documents needed for each individual participant were provided. At the start of the study each examiner had a nominal number of data collection sheets provided as well as electronic copies of all of the documents in the event that they needed more.

2.5 Dental Foundation Trainee support throughout data collection in practice

As well as training on the study day, support was provided throughout the data collection period to all the dental foundation trainees. On the study day, all the DFTDs were provided with a contact number and were advised to make contact at any point during the study for support or advice. A study WhatsApp group was created for all the DFTDs, so they could ask and benefit from general questions asked about the data collection process, no participant information was discussed in the WhatsApp group. Following the training day in Bristol, over the next 3 weeks, all the DFTDs were visited, at least once, in their individual dental practices, and where possible the DFTD's nurse, practice manager or educational supervisor, were met with to answer any questions they also had about the study and data collection. Throughout the data collection, I attended further practice visits where necessary and maintained regular contact with the DFTD's for updates and to provide motivation.

2.6 Data management

All data collected was anonymous, on enrolment the participants were allocated a study number which was written on the participant's inclusion and exclusion criteria confirmation, questionnaire and clinical form. I collected the data directly from the DFTD's, either at their individual dental practices or on their research presentation study days. On collection, all the data was reviewed and entries that could cause confusion were checked with the trainees and updated if required. The data was inputted by a specialist data input company (Adetiq, Brighton), all coded data was supplied to the study statistician, for analysis.

2.7 Statistical analysis

The data was transferred to SPSS (Version 23) for analysis. Preliminary, descriptive level analyses was performed for the questionnaire variables. For each clinical scoring variable, frequency distributions by site were produced and summarised by bar charts showing the variation between different areas of the mouth.

The main analyses were collected at a patient level. The relationship of each of the primary patient-level measures such as maximum recession depth at any site, or BEWE score of 2 or 3 was examined by cross tabulations to determine interrelationships between the measures. Potentially causal relationships were analysed by carefully selected multivariate regression analyses. Analysis established relative contributions of these risk factors.

All analyses have been characterised by suitable summary statistics with effect size measures (linear regression coefficient or odds ratio for logistic regression) with confidence intervals, as well as p-values.

Chapter 3: Results

Data collection took place between February 2017 and July 2017. The study recruited 814 participants from 28 sites, of which 60% were female and 40% were male. The 28 sites were located over the South West from Gloucestershire to Cornwall, the number of participants examined at each site varied depending on the site, shown in Table 3.1.

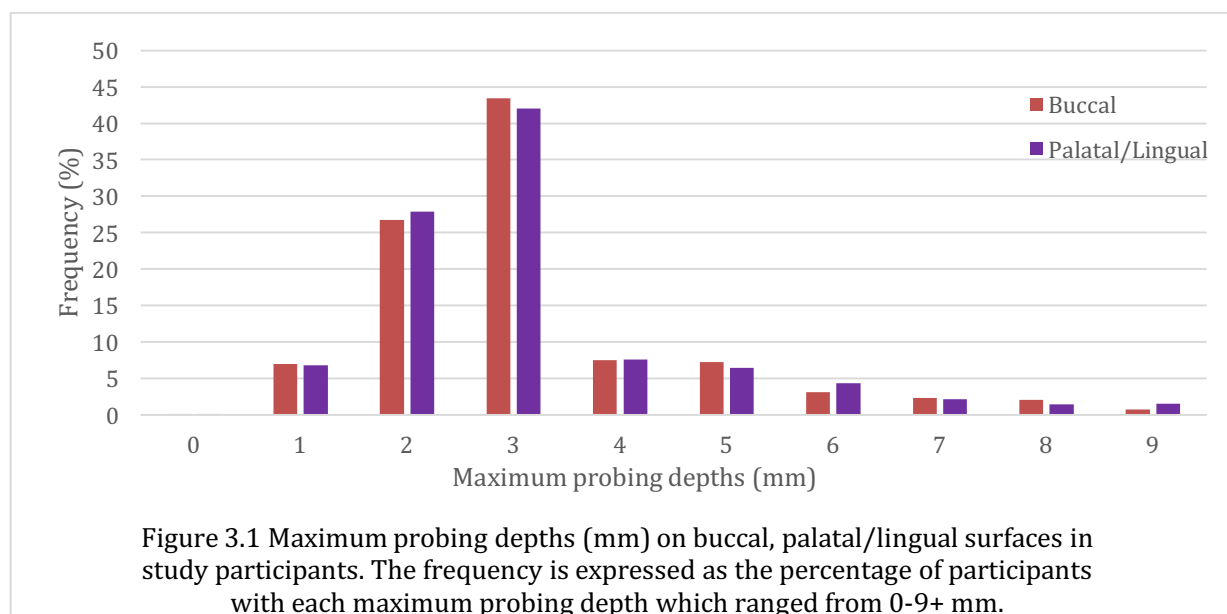
Table 3.1 The range in number of participants examined at individual sites

Number of participants from whom data was collected	Number of sites
0-10	2
11-20	5
21-30	9
31-40	5
41-50	1
51-60	2
61-70	-
71-80	-
81-90	-
91-100	2

3.1. Summary of clinical data

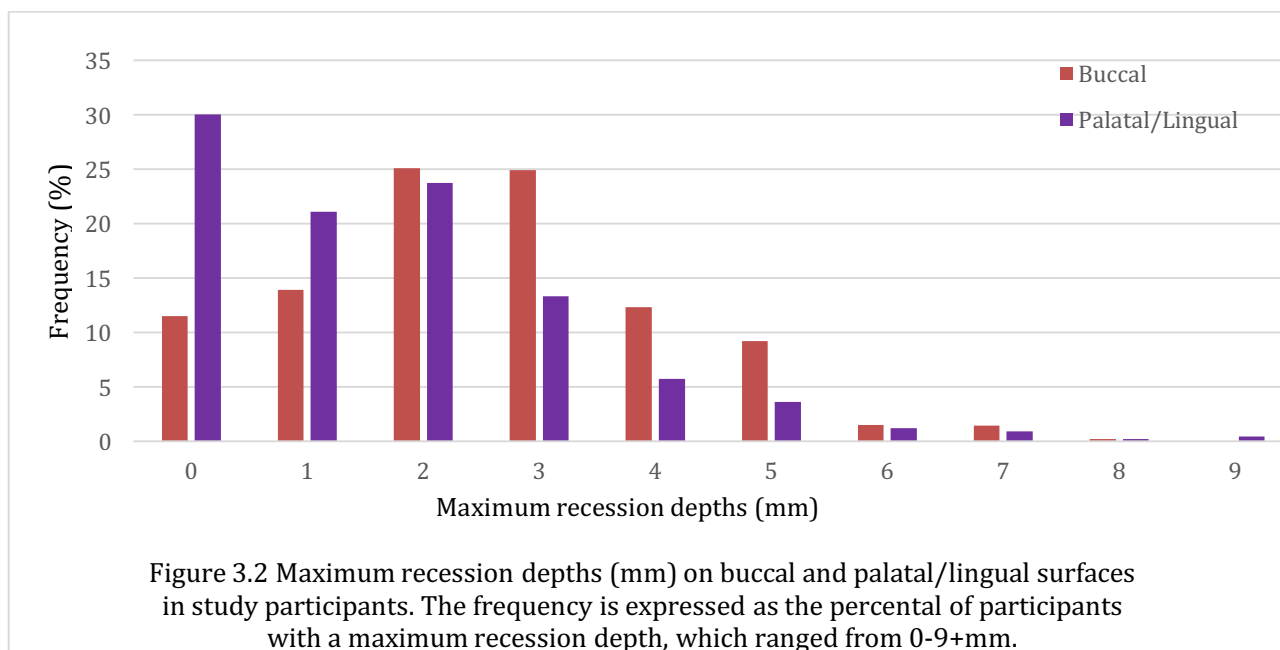
3.1.1 Periodontal scores

Participant maximum probing depths on buccal and palatal surfaces are shown in Figure 3.1, they ranged from 0-9mm, with maximum probing depths of 2mm or 3mm being the most common. Evidence of periodontitis was observed in 27.6% of participants who displayed pocket probing depth of 4mm or more, with 11% of participants displaying periodontal pocket probing depths of 6mm or more characterising severe periodontal disease.



Evidence of bleeding on probing as a marker of inflammation and active periodontal disease was observed in 75.6% of participants. Both the buccal and palatal/lingual surfaces showed similar percentage scores of bleeding on probing of 68.9% and 67.3%, respectively.

The maximum recession scores observed in participants are shown in Figure 3.2 and ranged from 0mm to 9mm+. Almost 90% of participants showed some evidence of recession with the most frequent maximum scores on the buccal surfaces being 2mm (25.1%) and 3mm (24.9%). Clinically relevant recession described as 3mm or more affected 49.5% of participants on the buccal surfaces and 25.2% of participants on the lingual/palatal surfaces. On the lingual/palatal surfaces, a score of 0mm, showing no recession was the most frequent score (30%).



3.1.2 Tooth wear and dentine hypersensitivity scores

Only 2.2% of all participants had a maximum BEWE score of 0, showing no detectable tooth wear in the participants dentition. Almost a quarter (24.4%) of participants showed evidence of only minimal tooth wear with a BEWE score of 1 on at least one site within their mouth, a score considered clinically acceptable. A maximum BEWE score of 2 or 3, representing clinically relevant tooth wear, was seen in 73.4% of participants.

Clinically relevant DH scores (Schiff 2 or 3), where the participant requested the clinician to stop and/or showed signs of discomfort to the cold air stimulus were seen in 23.9% of participants. Patient reported DH in response to a blast of cold air showed good agreement with the Schiff scores obtained ($\rho = 0.882$, $p < 0.001$). 74.8% of participants had dentine exposure on at least one tooth, and similarly to DH, dentine exposure was more common on the buccal compared to lingual/palatal surfaces.

3.2 Distribution of clinical conditions across the maxillary and mandibular dental arches

The distribution and the percentage contributions of clinical scores across the dental arch for each clinical variable scored for all participants with valid data are shown in Figure 3.3.

The frequency with which greater periodontal probing depths were recorded increased the more posterior the teeth were in the dental arch, probing depths of 3mm or more being most frequently observed on the molars. This trend was similar for all tooth surfaces in both dental arches. Similarly, there was a general trend that bleeding on probing occurred more frequently the more posterior in the mouth the teeth were, in both the maxillary and mandibular arches, for both the buccal and palatal/lingual surfaces. However, this trend was not as uniform as that observed for periodontal probing depths, as in the lingual and palatal data, towards the back of the mouth, bleeding on probing was more common around the first permanent molar as compared to the second permanent molar teeth.

Regarding recession, on the buccal aspect of the teeth, recession was observed most frequently on the maxillary canine, first premolars and first molar teeth as well as on the second premolar in the mandible. The frequency of recession on the palatal and lingual surfaces was lower than the buccal surfaces. Palatally, the surfaces mainly affected were the first molars, with the highest scores also being recorded on this tooth most frequently. By contrast, in the mandibular arch lingually, recession was observed most frequently with the highest frequency of scores of 3mm+ in the anterior region compared to the posterior premolars and molars. As expected, the distribution of dentine exposure closely matched the distribution of recession, again being more frequent on the buccal aspects of teeth compared to the palatal or lingual aspects.

The distribution of patient reported DH and clinically relevant DH, as measured by Schiff score (2 or 3), followed a similar pattern to both recession and the presence of dentine exposure buccally, in the maxilla, palatally and lingually, but buccally in the mandible frequencies were higher anteriorly. The overall frequency of clinically relevant DH, on palatal and lingual surfaces, however, was low.

On the buccal aspect of the teeth clinically relevant BEWE scores of 2 or 3 were observed most often in the canine, premolar and first molar region. Palatally and lingually, clinically relevant tooth wear was less common than buccal tooth wear and was more evenly distributed across the dental arches. BEWE scores of 2 or 3 were recorded most frequently in the incisor and canine region, lingually and palatally.

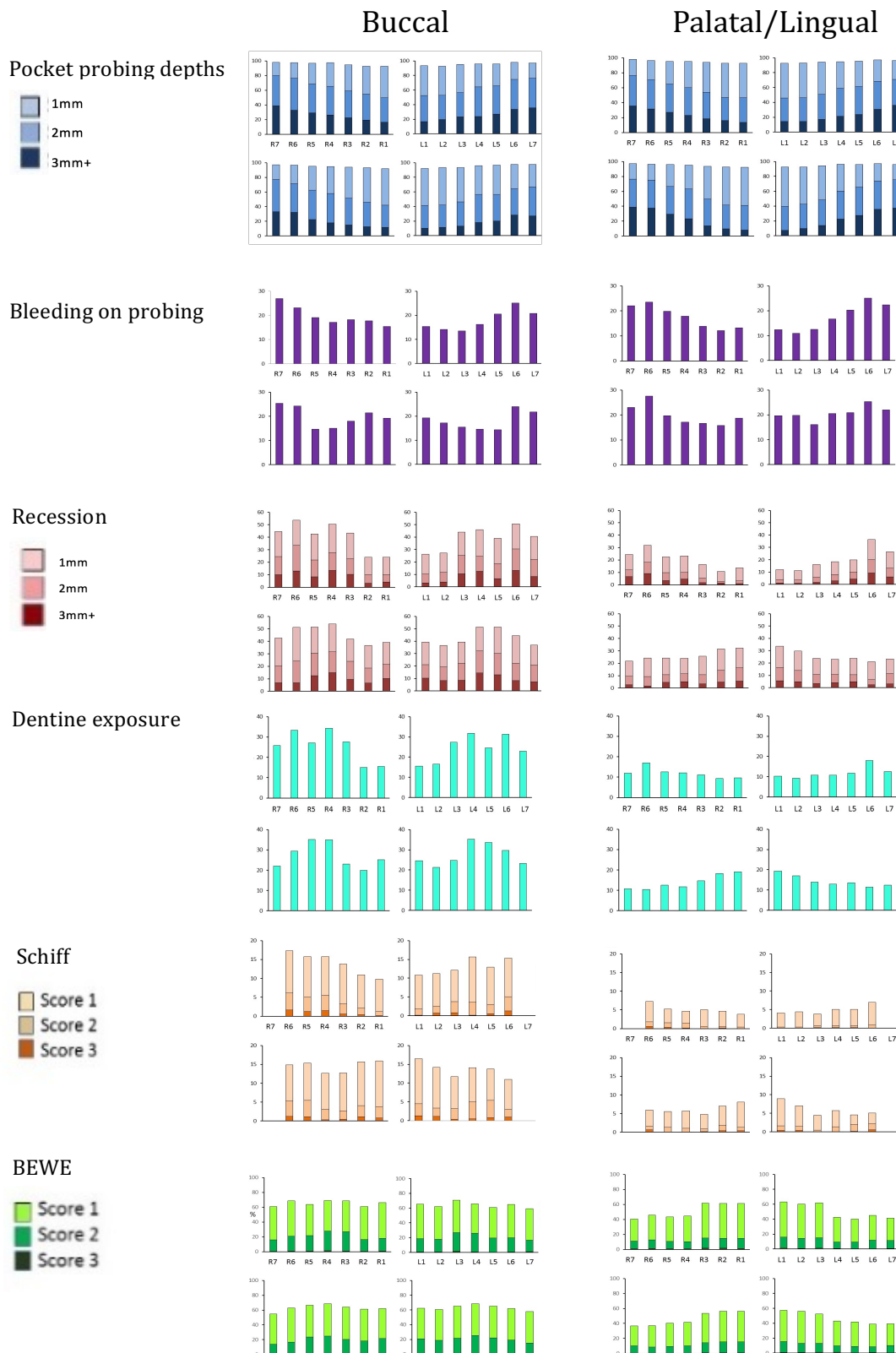


Figure 3.3 The distribution of clinical scores across the maxillary and mandibular arches on the buccal and palatal/lingual surfaces of teeth. The y axis of the graph represents the percentage of participants with this result once missing data had been removed from the analysis and the x axis of the graph represents the

3.3 Correlations between clinical conditions

Associations between periodontal measures and the other clinical conditions were determined, those that were the strongest are shown in Table 3.2.

A strong positive correlation was detected between the maximum probing depth and both the presence of bleeding, and maximum recession score.

Maximum recession score was also significantly positively associated with the presence of exposed dentine, and maximum tooth wear scores on the buccal and lingual/palatal surfaces. An inverse, yet strong correlation between tooth wear and bleeding was detected; showing that the more tooth wear there was the less bleeding was present and vice versa.

Table 3.2 Strong correlations detected between periodontal conditions and various other oral conditions

	Measure	95% CI	p value
Probing depth → Bleeding	U/mn* = 0.744	0.702-0.781	<0.001
Recession → Probing depth**	$\rho = 0.209$	0.142-0.274	<0.001
Dentine exposure → Recession	U/mn* = 0.804	0.767 - 0.830	<0.001
Tooth wear(B&L)→ Bleeding***	U/mn* = 0.395	0.350-0.441	<0.001
Tooth wear(B&L) → Recession**	$\rho = 0.409$	0.350-0.464	<0.001

*U/mn Generalised Mann Whitney measure (Mann Whitney U together with area under the curve)

**Spearman's Rank correlation

***Inverse correlation

In addition, strong positive associations were also identified between the maximum tooth wear score buccally and palatally/lingually and dentine exposure (U/mn=0.741, 95% CI 0.698-0.784, $p<0.001$) and as expected there was a strong positive correlation between dentine exposure and the presence of patient reported sensitivity (Odds Ratio 1.692, 95% CI 1.231-2.327).

No other strong associations between clinical conditions were detected.

3.4 Questionnaire descriptive statistics

3.4.1 Participant characteristics

Participants ranged in age from 18 to 91 years of age, with relatively similar numbers of participants in the 10-year age brackets from 21-30 to 61-70, Figure 3.4. Other general participant characteristics are outlined in Table 3.3.

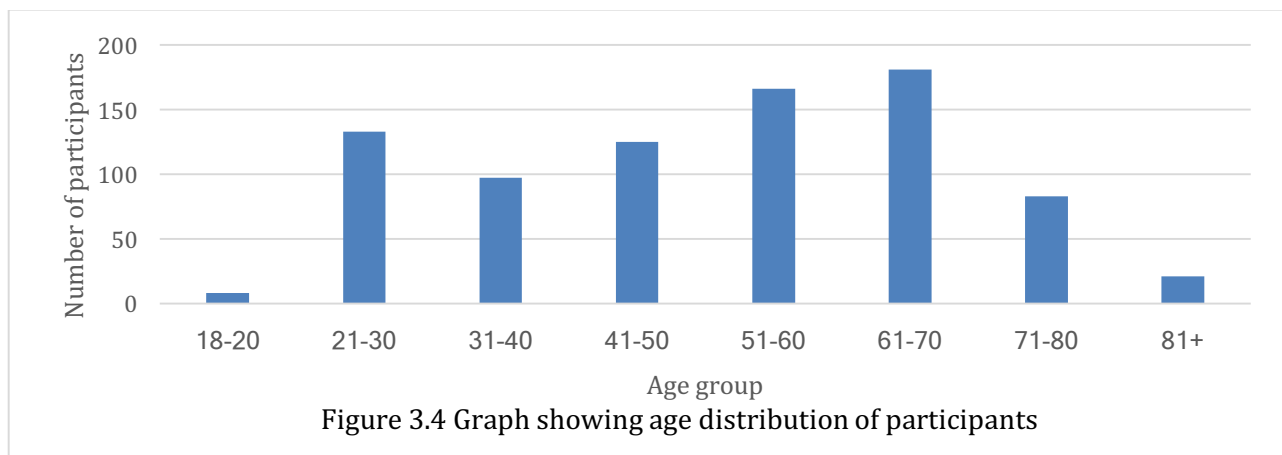


Table 3.3 General participant characteristics recorded from questionnaire

Variable		
Handedness	85% Right Handed (n=689)	15% Left Handed (n=123)
Mean height	168.88 cm	
Mean weight	74.98 kg	
Mean BMI	26.131	
Wearing dental braces	1.5% wore dental braces (n=12)	98.5% did not (n=788)
Wearing dentures	11% wore dentures (n=86)	89% did not (n=716)

3.4.2 Participant oral hygiene practices

Participant reported oral hygiene habits are shown in Figure 3.5 and were generally good. Data showed that 74% (n=602) of participants brushed their teeth twice a day, and there was a similar split of people tooth brushing before and after breakfast. Slightly more participants brushed most often with a powered (54%, n=439) as compared to a manual (46%, n=371) toothbrush.

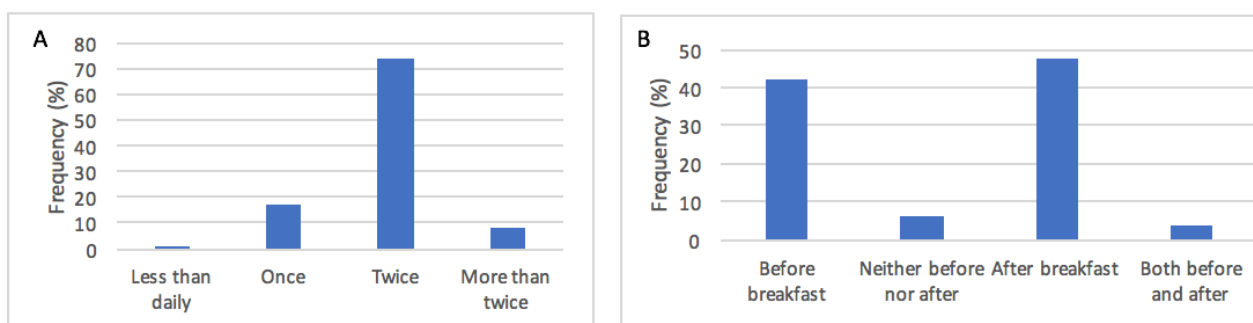


Figure 3.5 Participants brushing habits showing the number of times they reported brushing per day, including frequency (A) and timings of tooth-brushing with respect to breakfast (B).

Of the 800 participants who answered the question about whether or not they used a fluoride toothpaste 83% (n=665) stated 'yes', however, 118 (15%) did not know whether they did or did not. 117 (15%) participants also used fluoride in another form aside from toothpaste.

Dental attendance was good (Figure 3.6), the majority of participants had attended the dentist at least once in the 1 year prior to participating in the study, the number of visits to either the dentist or hygienist over the past year ranging from 0 to 10. Participants who had attended the dentist or hygienist had most commonly (67.2%, n=538) attended their last appointment for a check-up or clean.

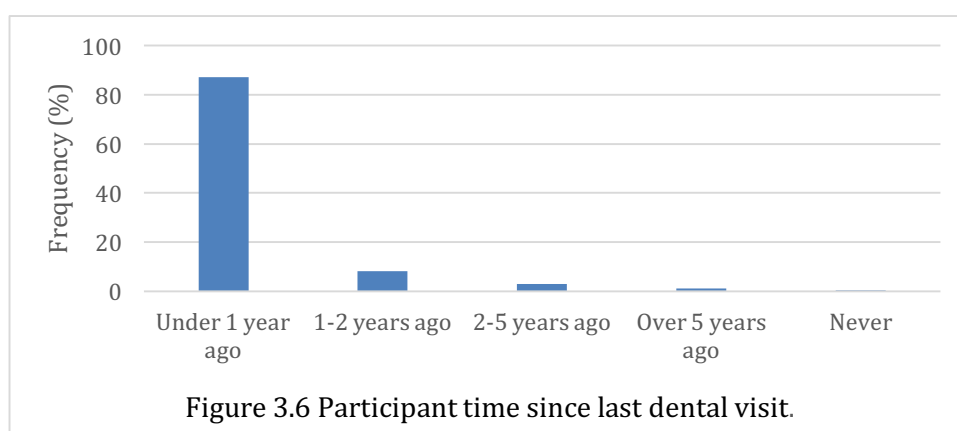
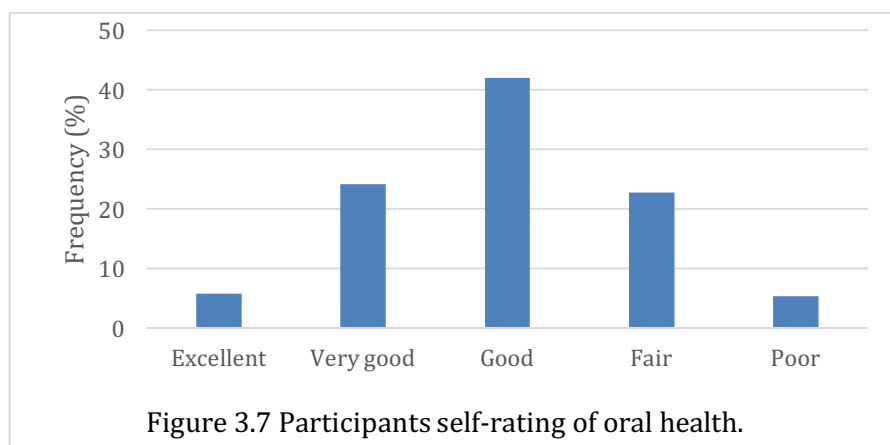


Figure 3.6 Participant time since last dental visit.

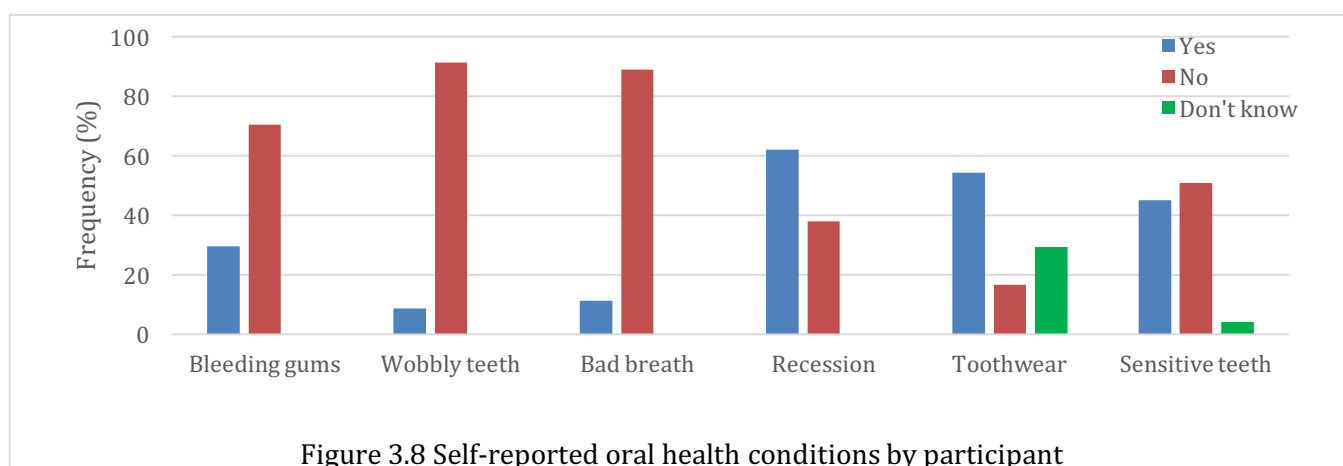
3.4.3 Participant reported oral health

Questions exploring patients' perceptions of their own oral health and whether they felt they suffered from oral problems indicated that the majority (42%, n=341) of participants rated their oral health as good, as shown in Figure 3.7.



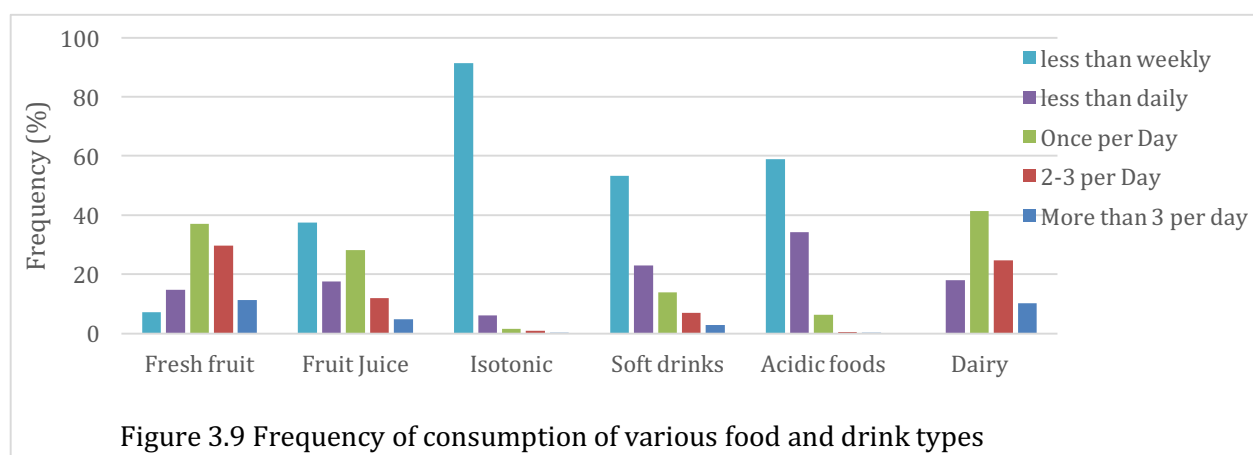
Participants were then asked about their experience of specific oral health conditions, (Figure 3.8). Of the 814 individuals who completed the questionnaire, 238 participants (29.5%) indicated their gums bled, of which 104 (44%) had used home treatments for bleeding gums and 155 (56%) had spoken to their dentist about their bleeding gums. 70 (8.8%) participants explained they had wobbly teeth and 87 (11.1%) participants indicated they had bad breath, of which 75 (87%) explained this had concerned them.

A large number of participants, 433 (54%) reported they had tooth wear, however 234 (29.3%) did not know whether they had tooth wear or not. Of the 433 participants who indicated they had tooth wear, 180 (42%) had talked to their dentist about their tooth wear. A large number of 360 participants (44%) reported they had sensitive teeth of which 241 (67%) had previously spoken to their dentist about their sensitive teeth. The majority of these participants (41.8%) had experienced sensitive teeth for over 5 years previously. Of the participants who reported sensitivity, the most common triggers for sensitivity were cold drinks (86.1%) and cold air (33.3%). The pain scores of sensitivities ranged from 1-10, but the most common pain scores were 2-5.

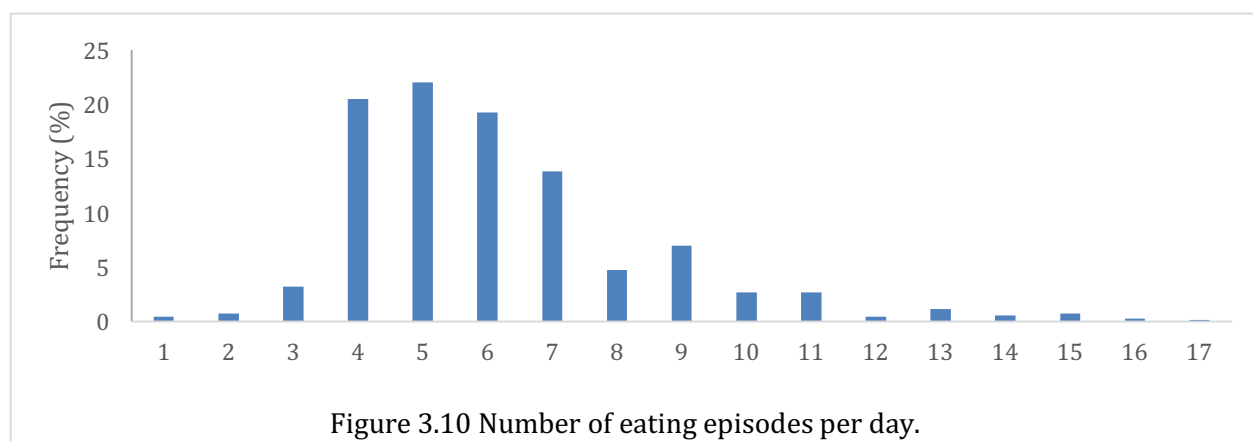


3.4.4 Participants experience of lifestyle risk factors

Responses to questions that asked about the participants diet are shown in Figure 3.9. The most commonly consumed acidic dietary product was fresh fruit, with 78% of participants eating fresh fruit at least once a day, while 45% of the participants consumed fruit juice and 24% of participants consumed soft drinks, at least once a day.



The number of eating and drinking episodes per day varied greatly, but was most commonly reported between 3 and 6, as shown in Figure 3.10.



Over 600 participants stated they drank alcohol and a large proportion of over 300 stated they snored. Other life style risk factors; such as smoking, reflux and stress were observed in some participants, shown in Figure 3.11.

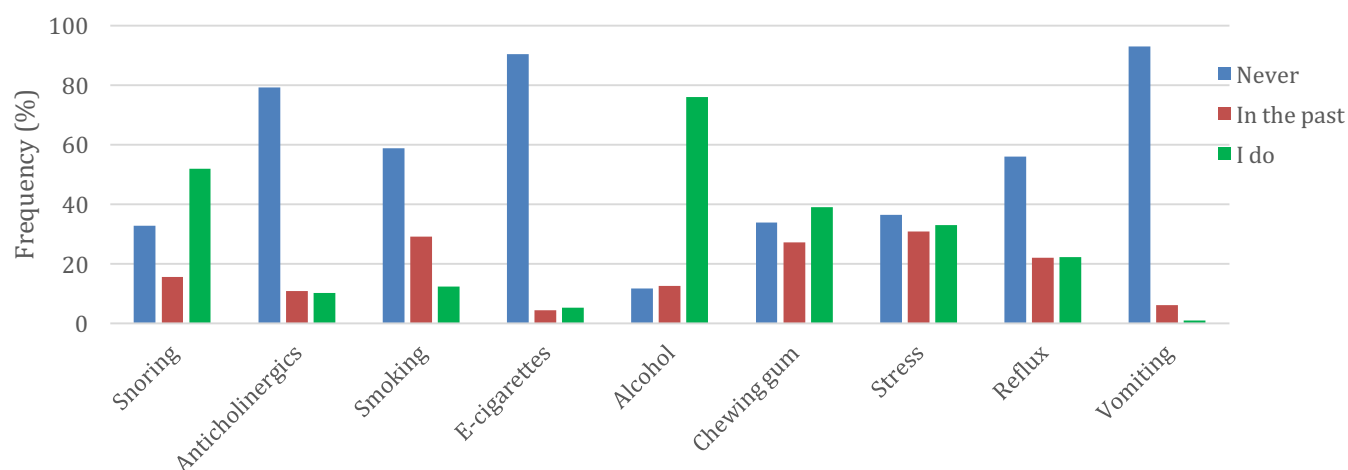


Figure 3.11 Oral health related lifestyle risk factors, participants selected whether they had been exposed to these factors currently, in the past or never at all

3.5 Associations between clinical periodontal findings and questionnaire variables

It was demonstrated that both recession and probing depth were strongly associated with age ($p = 0.511$, $p < 0.001$ and $p = 0.234$, $p < 0.001$, respectively). To eliminate the effect of age as a confounding factor an adjustment for age was made when determining the associations between recession or probing depth with potential risk factors that were also likely to be associated with age. Bleeding on probing was shown not to correlate with age ($p = -0.023$, $p = ns$), thus associations of potential risk factors with this variable were not age adjusted.

3.5.1 Associations between periodontal clinical scores and questionnaire variables that could be causal

Regarding periodontal probing depths, following age adjustments, in addition to age, only one other variable was significantly associated with periodontal probing depth of 4mm or more and could be causal for it, smoking (Table 3.4).

Table 3.4 Significant associations between the periodontal variable of periodontal probing depth of 4mm or more and questionnaire variables.

Association with probing depths of 4mm or more		
Variable	Comparison	p value
Age (4mm or more)	$\chi^2 = 38.57$	$p < 0.001$
Smoking (4mm or more)*	$\chi^2 = 32.98$	$p < 0.001$

*adjusted for age

The most important significant associations between bleeding on probing and questionnaire variables are shown in Table 3.5. Bleeding on probing was significantly higher in males compared to females, in those who were left handed and those who had not received any dental care in the preceding year.

Bleeding on probing was also strongly associated being overweight, the frequency of consumption of soft drinks, the number of eating and drinking episodes per day, smoking and the use of e-cigarettes, but the e-cigarette association could be secondary to smoking.

There was also a positive association between bleeding on probing and having seen a dentist for treatment for tooth wear ($\chi^2 = 4.12$, $p < 0.05$), the importance of sensitivity pain ($\chi^2 = 4.79$, $p < 0.05$) and a positive response for changing eating and drinking habits due to sensitivity (mean difference = 0.386, $p < 0.05$).

Table 3.5 Significant associations between the periodontal variable of bleeding on probing and associated questionnaire variables that may be causal for it.

Association with the presence of bleeding on probing		
Variable	Comparison	p value
Gender	$\chi^2 = 7.78$	$p < 0.01$
BMI (kg / m ²)	$t = 3.63$	$p < 0.001$
Brushing per day	$\chi^2 = 11.43$ (4 groups)	$p < 0.05$
Brushing hand (left)	$\chi^2 = 15.00$	$p < 0.001$
Smoking	χ^2 trend = 7.00	$p < 0.01$
Number of times eat/drink daily	χ^2 trend = 7.49	$p < 0.01$
Soft drinks	χ^2 trend = 6.13	$p < 0.05$
Time since previous dental visit	χ^2 trend = 5.00	$p < 0.05$

Regarding recession, after the data analysis was adjusted for age, in addition to age there were two significant relationships found. Firstly, smoking was potentially causal of clinically relevant recession (3mm or more) and the pain of DH was more important to participants who exhibited clinically relevant recession.

Significant associations between gingival recession and questionnaire variables are shown in Table 3.6. Thus, smoking was strongly associated with increased periodontal pocket probing depths of 4mm or more, an increased presence of bleeding on probing and an increased amount of recession of 3mm or more.

Table 3.6 Significant associations between the periodontal variable recession and associated questionnaire variables

Association with recession of 3mm or more		
Variable	Comparison	p value
Smoking	$\chi^2 = 8.67, 2 \text{ df (4 categories)}$	$p < 0.05$
Pain with DH	$\chi^2 = 16.33 \text{ 3 df}$	$p < 0.001$

Further analysis to explore the effect of handedness on periodontal conditions demonstrated that while maximum recession and maximum probing depths were significantly higher on the right compared to the left, yet this difference was not due to handedness.

3.5.2 Correlation between patient reported and clinician diagnosed oral conditions

Clinical periodontal variables were also correlated with patient reported oral health responses so see how closely they matched (Table 3.7).

Table 3.7 Significant associations of periodontal pocket probing depths, bleeding on probing and recession with patient reported oral health variables

Association with periodontal probing depth of 4mm or more		
Variable	Comparison	p value
Poor self-rated oral health	$\rho=0.259$	$p<0.001$
Bleeding on brushing	$U/mn = 0.652$	$p<0.001$
Wobbly teeth	$U/mn = 0.785$	$p<0.001$
Self-reported gum shrinkage	$U/mn = 0.604$	$p<0.001$
Self-reported tooth wear	K-W	$p<0.01$
Association with bleeding on probing		
Variable	Comparison	p value
Poor self-rated oral health	$\chi^2 \text{ trend}=18.47$	$p<0.001$
Bleeding on brushing	$\chi^2 =43.9$	$p<0.001$
Wobbly teeth	$\chi^2 =4.06$	$p<0.05$
Association with gingival recession		
Variable	Comparison	p value
Poor self-rated oral health	$\rho =0.158$	$p<0.001$
Wobbly teeth	$U/mn =0.734$	$p<0.001$
Self-reported gum shrinkage	$U/mn =0.693$	$p<0.001$
Self-reported tooth wear	K-W	$p<0.001$

The associations identified in table 3.7 indicated that study participants seemed to have a good general self-awareness of conditions present in their mouths which matched the findings of the clinical examination. For example, there was a strong association between self-reported bleeding on brushing and clinically determined bleeding on probing ($p<0.001$).

Chapter 4: Discussion

4.1 Participant demographics

This study recruited 814 adults aged 18 or over attending routine dental appointments at 28 NHS dental practices across the South West of England. This sample size is similar to the number recruited from the general population by mailshot of addresses selected at random in the South West in the latest UK ADHS carried out in 2009 (O'Sullivan *et al.*, 2011). In the ADHS in the South West region 1012 adults aged 16 or over were interviewed in 651 households, 663 participants also had a dental examination performed in their homes. The similarity of study size supports the importance of the findings of the study reported in this thesis.

In the study reported here, there was a 60:40 ratio of female to male participants. The higher proportion of female participants could be since females are more likely to attend routine dental examinations, compared to males (Fukai *et al.*, 1999). Interestingly, however, in general medical clinical trials there is often an underrepresentation of females (UHL 2007). The ages of the study participants ranged from 18 to 92 years old, and there was a relatively even distribution of participants across the age ranges up to age 70. UK population data from 2017 show approximately similar numbers of individuals at all ages up to 55, thereafter numbers decline steadily, although 18.2% of adults were still aged 65 or more (Park, 2018). While overall the present study age demographic was broadly in line with the UK population data, the percentage of participants aged 30-49 was slightly lower and the percentage aged 65+ was a little higher than the UK figures, this could be due to the working population having less time to participate in the study. This distribution of participants favouring those who are older is probably due to this age group having more time and thus being more willing to participate in the study. The mean height of the participants was 169cm (5,5") which, given the 60:40 female to male ratio is in line with UK figures, average male and female heights reported as 178cm (5'10") and 164cm (5,4"), respectively (Amos, 2016). The average BMI of the study participants was 26, with 37% overweight and 20% obese, figures that are similar to those reported in a national survey of UK adults over the age of 16 years old, which showed that 35% of the participants were overweight and 26% were obese (Obesity Statistics UK Parliament). In the current study, 11% of the participants wore dentures, a figure that is in line with that obtained in the 2009 ADHS, which found that 13% of dentate participants wore dentures in combination with their natural teeth (Steele *et al.*, 2011). The slightly lower percentage of patients wearing dentures in the present study, could be due to the fact that people with less than 10 teeth were excluded from participation, with more natural teeth there is less likelihood of needing a partial denture. Lastly, in this study, 85% of the participants were right handed and 15% left handed, this matches global figures for handedness (Balter, 2009).

Taken together, the data confirms that the participants in the study reported here are representative of the UK adult population, and that the data is comparable to the ADHS in many domains, including the sample size in the South West and participant demographics. The main difference between the present study and the ADHS is that people in this study were approached to participate whilst attending their regular NHS dental practice, while the ADHS selected people at random to participate using their postcode. In the ADHS, approximately one quarter of participants had not attended the dentist in the past 2 years (Steele *et al.*, 2011), thus the ADHS survey includes more data from non-regular dental attenders.

4.2 Prevalence of periodontal conditions

The present study investigated the prevalence of 3 conditions that are indicative of periodontal health or disease; pocket probing depth, the presence or absence of bleeding on probing and gingival recession depth. It was shown that just over a quarter of adults had evidence of periodontitis in at least one site in the mouth, a prevalence figure that is low compared to that reported in the last UK adult dental health survey, in which 45% of participants had evidence of periodontitis (White *et al.*, 2011). The difference in prevalence between the two studies could reflect a trend for improving periodontal health, as the periodontitis figure recorded by the ADHS fell by 10% between 1998 and 2009 (White *et al.*, 2011), although it is perhaps more likely that the lower figure found in the present study is a result of differences between the study populations, those in the ADHS did not necessarily attend the dentist. Given that in the present study just over a quarter of participants were aged 65 and yet only a quarter of all participants showed evidence of periodontitis, or over it appears that this group of NHS patients has better than average oral health.

Levels of severe periodontal disease in the current study were similar but slightly higher than those found in the most recent ADHS with 11% as compared to 8% of participants exhibiting periodontal pocket probing depths of 6mm or more. This prevalence figure in the current study is the same as the global figure reported by Kassebaum *et al* (2014). The fact that the severe periodontal prevalence figure was similar to that found globally, and reported in the ADHS 2009 and yet the figure for overall prevalence for periodontitis was low may be due to the participants in the study being regular dental attendees. In a cohort such as this with generally good oral health and oral hygiene habits, a lower incidence of new disease would be expected, yet at the same time, those with more severe disease attending the dentist regularly may have been able to maintain their periodontally compromised teeth for longer.

In contrast to the figures for periodontitis, the presence of bleeding on probing was substantially higher (75.6%) in the present study than that recorded in the ADHS (2009) (54%) (White *et al.*, 2011). However, in the ADHS only half the available tooth surfaces (buccal surfaces in the upper dental arch

and lingual surfaces in the lower dental arch) were examined, while in the present study bleeding on probing was recorded for all tooth surfaces in both dental arches, increasing the likelihood of detection. Furthermore, in the ADHS report it is acknowledged that in a field survey, where participants are examined in their own homes not in a dental chair in a clinic with good lighting, periodontal conditions such as bleeding on probing may not be obvious and thus figures are likely to underestimate the prevalence of conditions (White *et al.*, 2011). In comparison to gingival inflammation prevalence studies however, the figure for bleeding on probing recorded in the present study can be considered low, for example a multi-centre cross sectional study in South America showed 96.5% of adult participants had gingival inflammation (Carvajal *et al.*, 2016) and in a cross sectional study in Egypt gingivitis was detected in all four hundred and twenty five participants (Mostafa and El-Refai, 2018).

Gingival recession is multifactorial, and can be associated with both good and bad periodontal health (Kassab and Cohen, 2003). On one hand, recession is common in patients with periodontal disease, due to the pathological loss of alveolar bone supporting the teeth leading to exposed root surfaces (Kassab and Cohen, 2003). When recession is associated with disease it is more common around posterior teeth which are harder to clean, and in males who have been shown to have worse oral hygiene as compared to females (Mythri *et al.*, 2015, Seong *et al.*, 2018). This could be due to slowly progressing periodontal disease, formally known as chronic periodontitis, occurring more frequently in an older population whom may experience difficulty in maintaining excellent oral hygiene around their posterior teeth, regardless of the standard of professional advice and treatment they have received. However, gingival recession can also be associated with good oral hygiene practices, for example, secondary to overzealous tooth brushing (Addy and Hunter, 2003), this may also result in the trend of recession being found more frequently on buccal tooth surfaces (Seong *et al.*, 2018) (Albandar and Kingman, 1999). In the present study, the presence of clinically detectable gingival recession of at least 1mm was observed in almost 90% of participants. This figure is lower than that found in a recent cross sectional study of UK based young adults with minimal periodontitis, in which all study participants had evidence of recession on at least one tooth (Seong *et al.*, 2018), the recession observed likely a result of toothbrushing. Clinically relevant gingival recession of 3mm or more was observed in approximately half of the participants in the present study, by contrast, in a Greek study 63.9% of young adults had gingival recession of more than 3mm (Chrysanthakopoulos, 2014). Interestingly in the study by Chrysanthakopoulos (2014), recession correlated both with toothbrushing frequency and measures of poor oral hygiene (gingival inflammation/plaque/calculus), confirming its complex aetiology. In the present study, clinically relevant gingival recession was almost twice as common on buccal surfaces as compared to lingual/palatal surfaces, and this finding, together with the relatively low prevalence figures for periodontitis in this cohort suggest that perhaps the majority of the recession observed was associated with good oral hygiene practices.

In summary, the participants in this study appear to be health-conscious regular dental attendees, as they presented with a relatively low prevalence of periodontal disease and bleeding on probing. The relatively high prevalence of recession could be as a result of historic periodontal disease which has now improved or from overzealous tooth brushing, although no statistically significant association between oral hygiene practices and recession was detected as discussed below.

4.3 Patient reported oral health

The clinical findings from this study demonstrated that the oral health of participants was good, findings that were supported by their self-reported oral hygiene practices. The vast majority of participants brushed at least twice a day, most used a fluoride toothpaste and 15% indicated that they also used an additional source of fluoride. Overall, brushing frequency was slightly higher than that reported in the ADHS (2009), but the percentage of participants who confirmed their toothpaste contained fluoride was slightly lower (Steele *et al.*, 2011). Somewhat surprisingly almost all of those who did not positively indicate their toothpaste contained fluoride stated they didn't know if it did or not, by contrast, in the ADHS participants were able to provide a definitive answer to this question. However, given that the ADHS was undertaken at home, participants would have been able to confirm the fluoride content of their toothpaste, which likely accounts for the differences in findings. Participants in the present study were also regular dental attenders, the vast majority having attended within the last year, with appointments most commonly being for routine dental examinations or a clean. The participants of the most recent ADHS also most commonly attended their dentist for a routine check-up and two-thirds of the respondents attended the dentist regularly this appears to be representative of national dental attendance in England, an attendance survey carried out in 2016 discovered 56% of participants had seen an NHS dentist within the last two years and 15% mentioned they had a private dentist (NHS England, 2016). The oral hygiene described by the participants in the present study showed that the majority of them followed the recommended UK national guidance for dentists and patients, "Delivering Better Oral Health" which advises that all adults brush their teeth at least twice a day, with one brush always being last thing at night, with either a manual or powered toothbrush and a toothpaste containing at least 1350ppm fluoride. The lack of appointments for treatment indicates that the NHS practices from which participants were recruited for this study are providing good preventative advice regarding oral health care.

Given the good oral hygiene and regular dental attendance reported by study participants it might have been anticipated that they would be particularly dentally aware and that self-reported and clinical prevalence figures for specific indicators of periodontal disease would have been similar. However, bleeding on brushing was reported by only a third of study participants whereas clinically, bleeding on probing was present at at least one site in three quarters of participants. As almost half of

the participants had used home treatments for bleeding gums and just over half had spoken to their dentist about their bleeding gums it would appear that participants were aware that bleeding gums should be treated. The discrepancy in the findings could be due to bleeding being present only at the very back of the mouth where participants were not able to see it, or that bleeding was relatively minimal and less obvious when mixed with toothpaste and saliva as compared to what could be observed by a dentist with the patient sat in a dental chair under better light. Although there was a discrepancy in the amount of self-reported and clinically detected gingival bleeding, they were demonstrated to be significantly positively correlated. Similar findings, where self-reported bleeding of the gums was lower than clinically detected gingival bleeding have been found in other studies (Buhlin *et al.*, 2002, Mumghamba *et al.*, 2006), and in the study by Buhlin (2002), similar to the present study, the relationship between self-reported gum bleeding and that determined clinically was examined and found to be significantly associated. This suggests that while participants were not as able to detect gingival bleeding as their dentist, those with more bleeding were more aware of it.

In the present study, there was much closer agreement between the proportion of participants who reported wobbly teeth and the prevalence of severe periodontitis detected clinically. This could be due to the cumulative nature of periodontal disease, meaning the participants with periodontal disease, being regular dental attenders, were more likely to have been told by their dentist they have gum disease and about the sequelae of bone loss leading to mobile teeth over time. The presence of wobbly teeth and clinical periodontal disease were significantly positively correlated; periodontal disease was also correlated with self-reported bleeding on brushing. These findings suggest that the participants in the present study had a good awareness of their periodontitis. By contrast, when bone loss was detected by intraoral radiograph it did not correlate with a self-reported periodontal disease in those taking part in the Veterans Affairs longitudinal study (Pitiphat *et al.*, 2002), and in a second population reported in the same publication the authors demonstrated that self-reported periodontal disease was somewhat lower than that detected clinically. Although the responses to other questions regarding periodontal treatment were significantly correlated with clinical periodontal disease, Pitiphat and colleagues (2002) concluded that self-report was less accurate for periodontal disease than the other oral conditions they examined, and that improved self-report measures were needed for this oral condition. Similarly, it has been shown that there is a poor self-reporting of signs and symptoms associated with periodontal condition in the NHANES dataset (Liu *et al.*, 2010), with the biggest discrepancies in self-reporting and clinical findings in the older as well as less educated and lower income participants. Self-reported oral health has the potential to act as a useful tool in epidemiology studies (Blizniuk *et al.*, 2017), however, periodontal disease is usually painless and symptomless until the end stages (Ridgeway, 2000), therefore sufferers may not know that they have the condition, resulting in low self-reported figures. Therefore, for self-reported periodontal disease to be acceptable as a good indicator of clinical disease prevalence questions must be carefully designed and validated to

ensure that participants are able to answer them accurately. For example, a question which provides high sensitivity and specificity to the presence of periodontal pockets is when a person states that they have been told by their dentist or hygienist that they have 'deep pockets' (Blicher et al., 2005).

As indicated earlier, gingival recession may be a result of poor oral health combined with a periodontal condition or as a result of traumatic oral hygiene practices. In the present study, nearly two-thirds of participants reported their gums had shrunk or receded since they were younger, yet clinical examination showed that 90% of participants had some evidence of recession. This difference is likely to be because recession present may not be as visible to the participant in the mirror at home as compared what the dentist is able to see in a clinical exam in a dental chair, where lingual and palatal surfaces in particular are easier to visualise. Under reporting has been detected previously in a smaller study of 150 participants, where only 16.7% reported an awareness of gingival recession, even though over 60% showed generalised recession and tooth abrasion (Shetty et al., 2014), a discrepancy that is considerably larger than that found in the present study. Similar to the data for probing depths and gingival bleeding, although there were differences in prevalence figures for gingival recession, the presence of clinically relevant recession in the current study was significantly correlated self-reported gum shrinkage suggesting participant awareness of the condition. Interestingly clinically detected gingival recession was also significantly correlated with both self-reported wobbly teeth and tooth wear, the diversity of the correlations also supporting the idea that some recession present may be associated with poor oral hygiene and periodontal disease such as self-reported wobbly teeth and other areas of recession present may be associated with good oral hygiene where tooth wear is also detected.

While there were discrepancies in prevalence figures between specific clinical and self-reported periodontal measures, significant correlations were observed between them, and in addition, all three clinical measures of periodontal disease recorded in the present study also correlated with poor self-rated oral health. Thus, the data presented here indicates that the participants in this study are a group of health-conscious individuals, who follow good daily oral hygiene and regularly attend their NHS dentist who in the main, had a good awareness of their own oral conditions. The difference between self-reported oral health and clinical need in this population, is likely to be due to the participants reduced visibility to see their own teeth compared to a clinical examination.

4.4. Clinical correlations

It has been long established that as periodontal disease progresses, periodontal pockets become deeper due to loss of attachment and in active disease the inflammation presents as bleeding on probing (Lang *et al.*, 1986). Recession often occurs secondary to loss of attachment associated with periodontal disease (Kassab and Cohen, 2003), data shows there is a correlation between a dehiscence in supporting

alveolar bone and recession (Lost, 1984). Thus it would be expected that the periodontal measures examined clinically in this study would correlate with each other, and inversely with conditions such as tooth wear that have been associated with better oral hygiene (Savage *et al.*, 2018), which can be caused by increased tooth brushing frequency (Savage *et al.*, 2018) or combining tooth brushing with an erosive challenge (Addy and Hunter, 2003). However as indicated earlier, recession has a more complex aetiology (Addy and Hunter, 2003) and may be present in both healthy and diseased sites (Kassab and Cohen, 2003). Determining the relationships between clinical conditions is an important part of prevention of disease, it is important to understand how the treatment of/or advice for prevention of one oral condition may impact on another, a good understanding of the associations between oral conditions ultimately leading to better patient care and less disease.

In the current study, as anticipated, maximum probing depths were strongly correlated with the presence of bleeding on probing. Similar strong positive correlations between maximum probing depths and bleeding on probing have also been found in other recent studies. In a UK study of young adults, pocket probing depths were found to correlate strongly with both bleeding on probing and gingival recession (Seong *et al.*, 2018), and in a multicentre, cross sectional study in Germany, deeper periodontal pockets displayed a higher percentage of sites with bleeding on probing (Zimmermann *et al.*, 2015). The similarity between the distributions of higher probing depths and bleeding on probing in the dental arch observed in the present study supports this strong association, with both conditions occurring more frequently towards the back of the mouth. Other studies have also shown that posterior teeth have an increased tendency for periodontal disease and the associated features of periodontal breakdown such as bleeding on probing (Harris, 2003, Farina *et al.*, 2013). This finding could be due to reduced access to the posterior teeth for cleaning a suggestion supported by a recent study that found increased plaque scores on posterior teeth as compared to anterior teeth (Sreenivasan and Prasad, 2017), or due to a more complex crown and root anatomy of premolar and molar teeth compared to incisors and canines which has been associated with disease and reduced longevity (Hirschfeld and Wasserman, 1978).

The maximum periodontal probing depths in the current study were also strongly correlated with maximum gingival recession depths. This correlation was supported by the distribution of both measures in the dental arch where a similar pattern for the presence of maximum periodontal probing depths and maximum recession depths was observed, with each generally occurring least frequently the more anteriorly the teeth were in the mouth. The pattern of increased probing depths and recession occurring least frequently in anterior regions of the dental arch has also been reported recently in a recession prevalence study in young adults (Seong *et al.*, 2018) which in common with the present study demonstrated a significant positive correlation between gingival recession and periodontal pocketing. In the study reported in this thesis the highest recession scores were most commonly observed in the buccal premolar region of the maxillary teeth and lingual incisor region of the mandibular teeth. Previous studies have also demonstrated that gingival recession presents most frequently on the buccal aspect of tooth surfaces (Serino *et al.*, 1994) particularly in the premolar and

molar regions (Thomson *et al.*, 2000). The relationship between recession and periodontal pocket probing depths in this current study could be due to patients with previous periodontal disease, whom may have improved their oral hygiene and undergone treatment, so recession occurs, yet some diseased pockets persist.

Data suggests that the occurrence of recession in different areas of the mouth may be due to a variety of causes (Joshi *et al.*, 1994), recession present on premolars being likely due to traumatic brushing, whereas that occurring in the molar region being more likely to be associated with worse plaque control and a build-up of periodontal disease aggravating calculus deposits. In the present study, in addition to correlating with maximum probing depths, maximum recession scores also correlated with dentine exposure and maximum tooth wear scores. Furthermore, similar to recession depths, dentine exposure and higher tooth wear scores were seen more frequently buccally as compared to lingually and most commonly in the premolar region. It is common knowledge in periodontology, that when the gingivae recedes apically to the cemento-enamel junction and the root surface becomes exposed the root surface cementum is easily denuded to expose the underlying dentine, which may or may not be symptomatic (Seong *et al.*, 2018). Hence, it is unsurprising that maximum recession was strongly correlated with dentine exposure and their distribution in the dental arches was extremely similar.

Tooth wear is a prevalent condition which is multifactorial in nature (Milosevic, 1993). It has been shown that when tooth brushing and toothpaste are combined with an acid challenge, there is tooth wear to enamel and more severely to dentine (Hooper *et al.*, 2003). The participants in the present study were generally health conscious and the majority brushed twice a day, with approximately half of study participants brushing after breakfast, 80% drinking fruit juice at least once a day and just over a third reporting fruit juice consumption 3 or more times a day. Thus, it is possible that the correlation between recession and tooth wear observed in the present study was due to traumatic toothbrushing causing gingival recession (Addy and Hunter, 2003), and that toothbrushing alone or in combination with an erosive challenge such as orange juice then resulted in loss of the exposed dentine and DH. The localisation of greatest tooth wear and gingival recession to the buccal premolar region supports a role for abrasive tooth wear in the gingival recession observed on these teeth and the findings of Joshi *et al.*, (1994).

As would be predicted, tooth wear was inversely correlated with bleeding on probing, and the frequency of high tooth wear scores was generally lower on the posterior teeth in the dental arch while the bleeding on probing was overall most frequent in this area. This supports the idea that while this participant population had good oral health they brushed least effectively at the back of their mouths.

Taken together the correlations detected between clinical conditions in the present study, show that recession was detected most commonly on the buccal premolars this could be due to their position in

the dental arch combined with participants being more conscious about brushing the teeth which are on display and easier to access. Increased periodontal pocket probing depths and bleeding on probing in the posterior part of the mouth were seen across all the participants in the study, with both good and bad oral health, this identifies a need for better tooth brushing instruction by dentists around the back teeth and the development of more oral hygiene aids to improve cleaning in the posterior region.

4.5 Causal relationships

There are now some well-established risk factors for periodontal disease, including genetics (Michalowicz *et al.*, 1991), poor oral hygiene and smoking (Stabholz *et al.*, 2010). In addition, there are some emerging risk factors for periodontal disease, which are less well established, such as stress (Stabholz *et al.*, 2010) and diet (Jagannathachary and Kamaraj, 2010). As periodontal disease is a preventable disease with many negative impacts, such as tooth loss (Eaton and Ower, 2015), the risk of associated systemic disease (Nazir, 2017) and poor oral health quality of life (Ng and Leung, 2006) it is important to identify other possible causal risk factors. The study presented here collected data on both clinical conditions and by means of a questionnaire, details of demographics, lifestyle factors and oral hygiene practices, which when combined can help determine associations between individual's behaviours or characteristics and periodontal disease. While this study design cannot determine if particular factors are causal for periodontal disease, it can identify factors that should be investigated further.

Studies have shown an increased prevalence of periodontal pocket depths and gingival recession with age (Carvalho and Lussi, 2017, López *et al.*, 2017), this was supported by data in the present study, which identified that age strongly correlated with increased periodontal pocket probing depths and gingival recession. With age, there is a deterioration in immune function, making an older person more susceptible to periodontal disease and subsequently gingival recession (Preshaw *et al.*, 2004). Associations between gingival recession and periodontal pocket depths and questionnaire variable were therefore adjusted for age. Surprisingly, bleeding on probing was not correlated with age in the current study, this could be due to the reversible nature of gingivitis, whereby gingival health can improve with good oral hygiene at any point. The remaining analysis of the current study was adjusted for age.

In the current study, smoking was significantly associated with all three periodontal measures recorded, bleeding on probing, periodontal pocket probing depths and gingival recession. The current study agrees with evidence from previous studies that have led to the conclusion that smoking is a well-known and established risk factor for periodontal disease in a disease susceptible patient (Haber and Kent, 1992, Heitz-Mayfield, 2005, Zimmermann *et al.*, 2015), thought to be due to vasoconstriction of the periodontal tissue which negatively impacts wound healing and changes to the hosts inflammatory and

immune responses. With respect to gingival recession, while it is likely that the association with smoking is a result of the effect of tobacco use on the periodontium, given that gingival recession can also be caused by tooth brush abrasion it is also possible that some was caused by excessive tooth brushing with abrasion based pastes for whitening toothpastes by smokers attempting to remove stains associated with smoking. Interestingly, both smoking and the use of e-cigarettes, were associated with bleeding on probing, this is surprising as smoking is known to cause vasoconstriction of the gingival blood vessels and mask the effect of smoking on the periodontium preventing bleeding on probing (Farina *et al.*, 2013). The association observed in the present study may be due to the study including a combination of both heavy and light smoker, of which the light smokers experienced less disease masking.

Being male also correlated with poor periodontal health, as in the current study males had significantly more bleeding on probing compared to females. This study findings agree with current literature that periodontal disease is more common in males (Susin *et al.*, 2005, Haytac *et al.*, 2013, Eke *et al.*, 2015). The reasons for these findings are likely to be due to males having worse oral hygiene and plaque control compared to females (Furuta *et al.*, 2011) or a genetic variation in immune function which increases the risk of periodontal disease in males (Shiau and Reynolds, 2010).

Previous studies have associated obesity with poor oral hygiene and gingivitis (Franchini *et al.*, 2011, Martinez-Herrera *et al.*, 2017) and a recent study has suggested it is as an independent risk factor for periodontal conditions (Suvan *et al.*, 2018), this was supported by data in the current study which showed that bleeding on probing was strongly associated with being overweight. The frequency of consumption of soft drinks and the number of eating and drinking episodes per day in the current study were also strongly associated with bleeding on probing, this is unsurprising, as plaque on the tooth surfaces is formed from food, saliva and bacteria (Eaton and Ower, 2015) and its presence at the gingival margin leads to gingivitis which presents as bleeding on probing. The association between obesity and periodontal diseases including gingivitis, has been found to be due to obesity causing an exaggerated inflammatory response or an increased production of reactive oxygen species (Martinez-Herrera *et al.*, 2017), enabling the initiation and progression of periodontal diseases.

Poor oral hygiene is a well-known and established risk factor for gingivitis (Löe *et al.*, 1965), and this study confirms the finding that irregular plaque removal can lead to gingivitis presented as bleeding on probing, with those participants who brushed the least having the most bleeding on probing. In addition, participants in the present study who had not received any dental care in the preceding year were found to have an increased presence of bleeding on probing, this is similar to data reported in the 2009 ADHS which showed poor dental attendees also had more bleeding on probing (Hill *et al.*, 2013), this is likely to be due to regular attenders receiving repeated and up to date advice on how to maintain the health of their teeth and gums. Surprisingly, bleeding on probing was also associated with left handedness, this is interesting as the evidence on whether handedness influences a patient's oral hygiene is

controversial and inconclusive, some studies have identified that left handed people have worse oral hygiene (Cakur *et al.*, 2011) whereas others have shown better oral hygiene (Tezel *et al.*, 2001) due to left handedness being associated with an intrinsic neurological advantage.

Lastly, bleeding on probing was found to be positively associated with having seen a dentist for treatment of tooth wear, the importance of sensitivity pain and a need of changing eating and drinking habits due to sensitivity. The reasons for these associations are not immediately obvious, bleeding on probing although minimal could be caused secondary trauma caused by harsh brushing, but more investigations would be required to confirm this.

In conclusion, the potentially causal variables associated with increased periodontal pocket probing depths, bleeding on probing and clinically relevant recession in the current study, support current research and knowledge that smoking, being male and obesity are risk factors of periodontal disease. Again, the study findings show that participants in the current study are generally representative of the UK adult population.

4.6 Study design and limitations

The purpose of this cross-sectional multicentre epidemiology study was to assess and obtain accurate prevalence figures for periodontal disease in the South West of England. A study of this magnitude has not been carried out since the last ADHS in 2009, almost 9 years prior to the present study. Information about other oral conditions was recorded, so the study was able to determine the association of periodontal disease with other oral conditions and identify potential risk factors. The study also gave DFTDs an experience of undertaking dental research, an opportunity they would not usually have had as an undergraduate dental student or whilst working in general dental practice. The study, which included 814 participants, was large which made it possible to compare it to other epidemiological studies and had minimal inclusion and exclusion criteria, meaning as representative sample of people attending NHS dental practices as possible were able to partake in the study.

At the training day held at Bristol Dental Hospital, the DFTDs were taught how to recruit participants and carry out the study in their practices, so at the end of the day they left with the knowledge and skills needed to roll out the study in their practices. The DFTDs were trained on how to recruit participants, complete the necessary paperwork and in the afternoon, there was a hands-on clinical session. The hands-on session was very beneficial for introducing the data collectors to the clinical forms, and providing training in how to use the BEWE, which some of them had not used before. After the training day, the DFTD's were supported in practice, and had individual practice visits to support them with data collection. There was a WhatsApp group created for all the data collectors, where the members could ask questions and they could all learn from the answers as well as motivate each other. All of the data was double-checked when it was received at Bristol Dental School prior to being forwarded to the data inputters for statistical analysis. This involved making sure the participant ID and site ID was written

on each page, the correct consent was obtained, the questionnaires were clearly marked, and the clinical forms were clear and complete. Finally, the data collected was very thorough, using a full mouth approach and collecting multiple periodontal and tooth wear scores, this clinical data together with the lengthy questionnaire, enabled a clear picture of the population.

To make the project as straight forward and easy to perform as possible for the DFTDs, each site was provided with 20 printed copies of the master documents to get them started with data collection. For DFTDs who required more copies a USB stick was provided containing the study documents as word documents, unfortunately in a couple of cases, when these documents were printed from the sticks there were some formatting errors which affected some of the data recording sheets. This problem was only recognised when the data sheets were being checked prior to data input, however it was possible to identify issues arising from the altered formatting and upload the data, so that the results were not affected, but in the future master copies of documents will be supplied as PDFs.

Feedback from the DFTDs at the end of the study, indicated that they found data collection lengthy, as the questionnaire was long and there were lots of clinical parameters to examine and record, which made it difficult for them to complete alongside treating their patients and completing other necessary tasks in their training year. Data collection may have taken longer for these dentists compared to dentists with more clinical experience, and in hindsight this should have been accounted for, with time built in to their daily schedule for study data collection. In addition, even though the DFTDs went through an intensive study initiation day, which included Good Clinical Practice (GCP) training, it became apparent at the time of data checking at the end of the study that one DFTD had not understood just how important the principals of GCP are. The majority of the consent forms collected by this DFTD bore the same signature, and when asked the DFTD explained that they had completed them after the participant had left as there had been so many sheets to fill out the consent forms had been missed. These participants had given verbal informed consent to take part however without valid written consent, their data had to be excluded from this study. Perhaps inviting the dentists nurse to the training day would have been beneficial as the nurse plays a crucial role in record keeping, they would then also have been trained in the absolute need for written informed consent and how to correctly fill in the dental charts, in addition having the dental nurse on board may have improved the DFTD's motivation towards the project.

An aspect of data collection which was not recorded accurately across most the practices, was the log sheet of the participants who declined to take part in the study with a reason for their choice to decline. This may have been poorly recorded due to the participants being approached to take part by receptionists, whom are often busy and may not have understood the importance of collecting this information. It would have been interesting to identify the number of participants who decline to take part in the study and their reasons for refusal, in hindsight I would have extended the training to include receptionists as well when I visited the practices.

Overall the study was a success, the project provided a research introduction and opportunity for young, newly qualified dentists to gain an experience that would not usually be available to them as undergraduates or general dental practitioners working in general practice. The young dentists played a huge role in collecting the data, and through having multiple sites and working as a team, their efforts produced a good-sized dataset which was comparable to the other large cross-sectional studies such as the ADHS. The study design was suitable to collect the data required to make an assessment of the prevalence of periodontal disease, its relationship with other oral conditions and identify potential risk factors. In hindsight, it would have been interesting to incorporate a medical history in the questionnaire to assess the association of any diseases with periodontal disease, however the questionnaire was lengthy enough, so there is good reason as to why it was not included in this study.

Chapter 5: Conclusion

In conclusion, this study observed that in this population of NHS patients, prevalence of periodontal disease was relatively low in comparison to the most recent ADHS and figures for other European nations. Self-reported oral hygiene measures were also suggestive of a cohort of patients who were dentally aware with good self-performed oral hygiene and effective, professional oral hygiene advice and treatment.

Significant strong associations were identified between periodontal pocket probing depths and bleeding on probing and periodontal pocket probing depths and gingival recession, as well as between tooth wear and gingival recession. Clinically relevant recession being associated with both tooth wear and periodontitis, supports a dual aetiology for gingival recession, whereby it can arise as a result of both good and poor oral hygiene practices.

The potential causal risk factors of periodontal disease in the present study included smoking and gender. Smoking was associated with all three periodontal parameters, periodontal pocket probing depths and gingival recession, whereas gender was associated with periodontal probing depths and bleeding on probing. A potentially causal, risk factor identified was obesity, but when the data was age adjusted this was identified as being less significant.

The findings from this study will impact professionals, as it has identified that well looked after mouths have a low incidence of periodontal disease. The data will have an impact on patients, as it supports evidence that smoking and being male are risk factors for periodontal disease, and prevention is the best measure to reduce disease occurrence.

This study is beneficial to the public, as it reinforced the message that regular dental attendance and effective self-directed and professional oral hygiene can lead to healthy and disease-free mouths, which is beneficial to the NHS and demonstrates the potential benefits of NHS dentistry. Future work would include performing this study on a larger scale, to compare different UK populations and establish clinical need over various locations. This could potentially be carried out at repeated time frames, such as the ADHS, which reviews trends and changes in oral health care and the prevalence of disease.

This study has initiated an interest for me in the link between periodontal disease, obesity and diet, and it would be interesting to explore further the effect of a high carbohydrate diet on bleeding on probing and periodontal pocket probing depths. This would have its own challenges as periodontal disease is a cumulative disease which occurs overtime, yet eating habits and preferences can change throughout life.

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Appendices

Appendix 1 – Study questionnaire

Site code
Participant ID

DF1: Questionnaire

Questionnaire

1. Are you: | Male ☐ Female ☐ (Please tick one box only)
2. What is your date of birth
D D M M Y Y Y Y
3. Please enter your age (in years) when you left full time education
4. How many times per day do you brush your teeth? (Please tick one box only)
- Less than once a day ☐
Once a day ☐
Twice a day ☐
More than twice a day ☐
5. Which kind of toothbrush do you use more often? (Please tick one box only)
- Manual ☐ Power (Electric) ☐
6. Do you usually brush your teeth (Please tick one box only)
- Before breakfast ☐ Neither before or after breakfast ☐ After breakfast ☐ Both before and after breakfast ☐
- IF you brush after Breakfast:
(6a) How long do you wait before brushing your teeth after having your breakfast? (Please give estimated average)
Number of minutes
Now go to question 7
- Go to question 7
7. Are you right-handed or left-handed? (Please tick one box only)
- Right-handed ☐ left handed ☐
8. How would you rate your oral health? (Please tick)
- Excellent ☐ Very good ☐ ~~Good~~ ☐ Fair ☐ Poor ☐
9. Do your gums bleed when you brush your teeth? (Please tick)
- Yes ☐ No ☐
10. Have you used anything for your bleeding gums to try to treat them yourself at home? (Please tick)
- Yes ☐ No ☐
11. Has your dentist talked to you about/treated/given you any treatment for your bleeding gums? (Please tick)
- Yes ☐ No ☐

Site code
Participant ID

DF1: Questionnaire

12. Do you have any wobbly teeth?

Yes ☐

No ☐

Go straight to question 14



13. Do your wobbly teeth affect you when you are eating?

Yes ☐

No ☐

14. Do you think you have bad breath?

Yes ☐

No ☐

Go straight to question 16



15. Do you worry about your bad breath?

Yes ☐

No ☐

16. Have your gums shrunk or receded? Can you see more of your tooth than you could when you were younger?

Yes ☐

No ☐

17. Do you think your teeth are showing signs of toothwear? *(Please tick)*

Yes ☐

No ☐

Don't know ☐

Go straight to question 20



18. Have you used anything for your toothwear to try to treat it yourself at home? *(Please tick)*

Yes ☐

No ☐

19. Has your dentist talked to you about/treated/given you any treatment for your toothwear? *(Please tick)*

Yes ☐

No ☐

20. Do you suffer from sensitive teeth? *(Please tick)*

Yes ☐

No ☐

Don't know ☐

Please go to the next page and answer question 28



21. Have you used anything for your sensitive teeth to try to treat them yourself at home? *(Please tick)*

Yes ☐

No ☐

22. Has your dentist talked to you about/treated/given you any treatment for your sensitive teeth? *(Please tick)*

Yes ☐

No ☐

23. How long have you been suffering from sensitive teeth? *(Please tick)*

Less than a year

☐

1 year – 1 year 11 months

☐

2 to 4 years 11 months

☐

5 or more years

☐

Don't know

☐

Site code
Participant ID

DF1: Questionnaire

24. What triggers the pain of sensitive teeth occur? *(Please tick all that apply, or if 'other' please describe)*

Toothbrushing ☐ Touch ☐ Hot drinks ☐ Sweet ☐ Cold weather (air) ☐ Cold drinks/ice ☐
Other

25. How painful are your sensitive teeth?

(please circle the number below to indicate how painful your sensitive teeth are)

No pain 1 2 3 4 5 6 7 8 9 10 Worst pain imaginable

26. Could you indicate which teeth are sensitive, so that they can be marked on the chart below (a member of the study site will help you fill this in)

upper left	7 6 5 4 3 2 1	1 2 3 4 5 6 7	upper right
	7 6 5 4 3 2 1	1 2 3 4 5 6 7	
lower left			lower right

27. How would you evaluate the pain intensity of your sensitive teeth? *(Please tick one box only)*

Not important ☐ = Little importance ☐ Some importance ☐ Very important ☐ Don't know ☐

28. This question is about any pain or sensation from your teeth – please answer even if you do not consider your teeth to be sensitive:

Thinking about yourself over the last month, to what extent would you agree or disagree with the following statements

(please tick one answer per question)

	Strongly Disagree	Disagree	Disagree a little	Neither agree or disagree	Agree a little	Agree	Strongly agree
Having sensations in my teeth takes a lot of the pleasure out of eating and drinking							
It takes a long time to finish some foods and drinks because of sensations in my teeth							
I have to change the way I eat or drink certain things							
I have to be careful how I breathe on a cold day							

Site code
Participant ID

DF1: Questionnaire

29. Do you.....? (Please tick, one answer per line)

	Yes I do	I have in the past	Never	Don't know
Snore				
Take sleeping medication/antidepressants				
Smoke tobacco (cigarettes, cigars, pipe)				
Smoke e-cigarettes				
Drink Alcohol				
Chew gum				
Suffer from stress				
Suffer from heartburn/reflux/regurgitation				
Suffer from repeat vomiting				

30. How many eating/drinking occasions do you have per day even in small quantities? Number of times

31. How often in the last month have you eaten or drunk the following, even in small quantities?

	More than 3 times per day	2-3 times per day	Once per day	Less than once per day, but at least once a week	Less than once per week
Fresh fruit eg orange, apple, grapes					
Fruit or vegetable juice eg orange, carrot					
Isotonic drinks/energy drinks eg poweraid, red bull					
Soft drinks eg cola, lemonade, squash					
Acidic food (not fruit), eg pickles					
Cheese, yoghurts or other dairy products					

32. Before today, when did you last visit a dentist or hygienist about your teeth, dentures or gums? (Please tick one box only)

Less than a year ☐ 1 year – 1 year 11 months ☐ 2 to 4 years 11 months ☐ 5 or more years ☐
Don't know ☐

33. How many times in the past 12 months have you seen a dentist or hygienist? Number of times

Site code
Participant ID

DF1: Questionnaire

34. What was the reason for your last visit to the dentist or hygienist? *(Please tick one box only)*

Check-up, examination or cleaning ☐

Routine treatment ☐

Emergency treatment ☐

35. How tall are you? *(Please give your answer in cm)* cm

36. How much do you weigh *(Please give your answer in kg)* kg

37. How often do you exercise or play sport? *(Please tick one box only)*

5 times a week or more ☐

3 to 4 times a week ☐

1 to 2 times a week ☐

1 to 3 times a month ☐

Less often ☐

Never ☐

38. Do you have dental braces

Yes ☐

No ☐

39. Do you have any removable teeth (dentures)

Yes ☐

No ☐

40. Does the toothpaste you usually use contain fluoride?

Yes ☐

No ☐

Don't know ☐

41. Do you use fluoride in any other form than toothpaste?

Yes ☐

No ☐

Don't know ☐

Appendix 2 – Study clinical forms

IRAS ID: 218303, Version 1.0, 25th Oct 2016

Clinical Form – page 2 of 2 (BEWE code and dentine exposure)

Site ID

Assessment Date

Participant ID

DoB

Please indicate if participant has a Dry Mouth
 No ☐ Yes ☐

BEWE codes [Bartlett, Gauss, Lussi, 2008] modified per tooth and on the buccal/palatal and occlusal surfaces

0. No erosive wear

1. Initial loss of surface texture

2. Distinct defect, hard tissue loss minor, less than 50% of surface area (clinical crown)

3. Hard tissue loss more or equal than 50% of the surface area (clinical crown)

Localisation codes

0. Anatomical crown and over cervical localisation

1. Anatomical crown in cervical localisation

2. Anatomical root in cervical localisation

3. Anatomical crown and root cervical localisation

Dentine Exposure

Dentine exposed:

0. No

1. Yes

BEWE + localisation codes + dentine exposure	Buccal	Severity																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																											</
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For all conditions: U. Cannot be determined X. Excluded or missing teeth

Clinical form page 1 of 2 (periodontal conditions and hypersensitivity)

Site ID		Participant ID					
Assessment	<input type="checkbox"/> D	<input type="checkbox"/> W	<input type="checkbox"/> M	/	<input type="checkbox"/> Y	<input type="checkbox"/> Y	<input type="checkbox"/> Y
Date							
DOB	<input type="checkbox"/> D	<input type="checkbox"/> W	<input type="checkbox"/> M	/	<input type="checkbox"/> Y	<input type="checkbox"/> Y	<input type="checkbox"/> Y

BPE:

0. No pockets >3.5mm, no calculus/overhangs
no bleeding on probing

1. No pockets >3.5mm, no calculus/overhangs
+ve for bleeding on probing

2. No pockets >3.5mm, +ve for supra or sub
gingival calculus/overhangs

3. Probing depth 3.5-5.5mm

4. Probing depth >5.5mm

► Furcation involvement
(*please include score too*)

S	f	S	S	f
S	f	S	S	f

Periodontal conditions

Gingival Biotype:

- Thick: probe under gingival margin = pink
- Thin: probe under gingival margin = grey

Probing depth:

1-9 mm (S₉: record as 9+)

Recession (CEI to gingival crest):

1-9 mm (S₉: record as 9+)

False pockets should be recorded in mm but as negative values

Bleeding:

1. Yes 0. No

Dentine hypersensitivity index

1. Yes 0. No

SCHIFF

- Subject does not respond to air stimulus
- Subject response to air stimulus but does not request discontinuation of stimulus
- Subject responds to air stimulus and requests discontinuation of stimulus
- Subject responds to air stimulus, considers stimulus to be painful and requests discontinuation of stimulus

[illegible]

For all conditions: U. Cannot be determined X. Excluded or missing teeth